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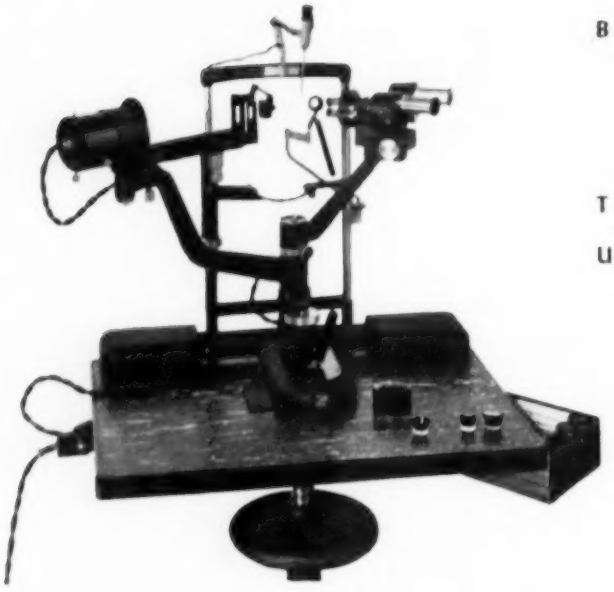

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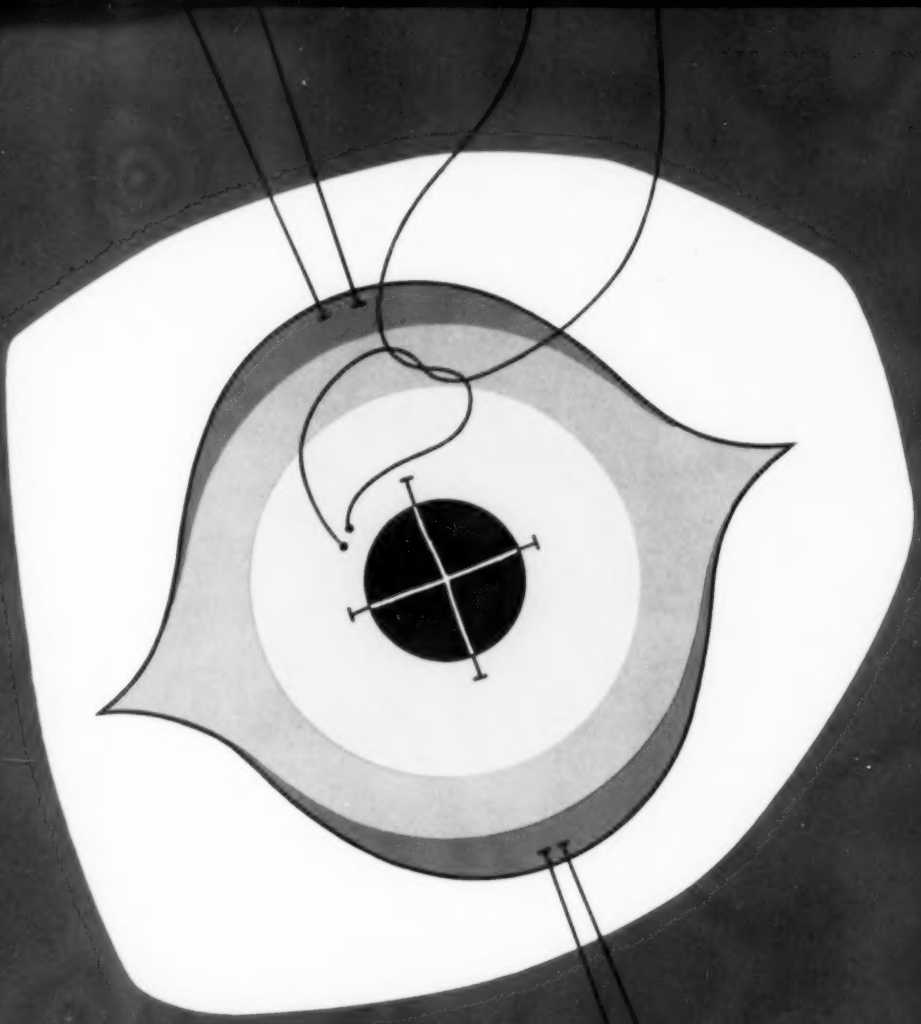

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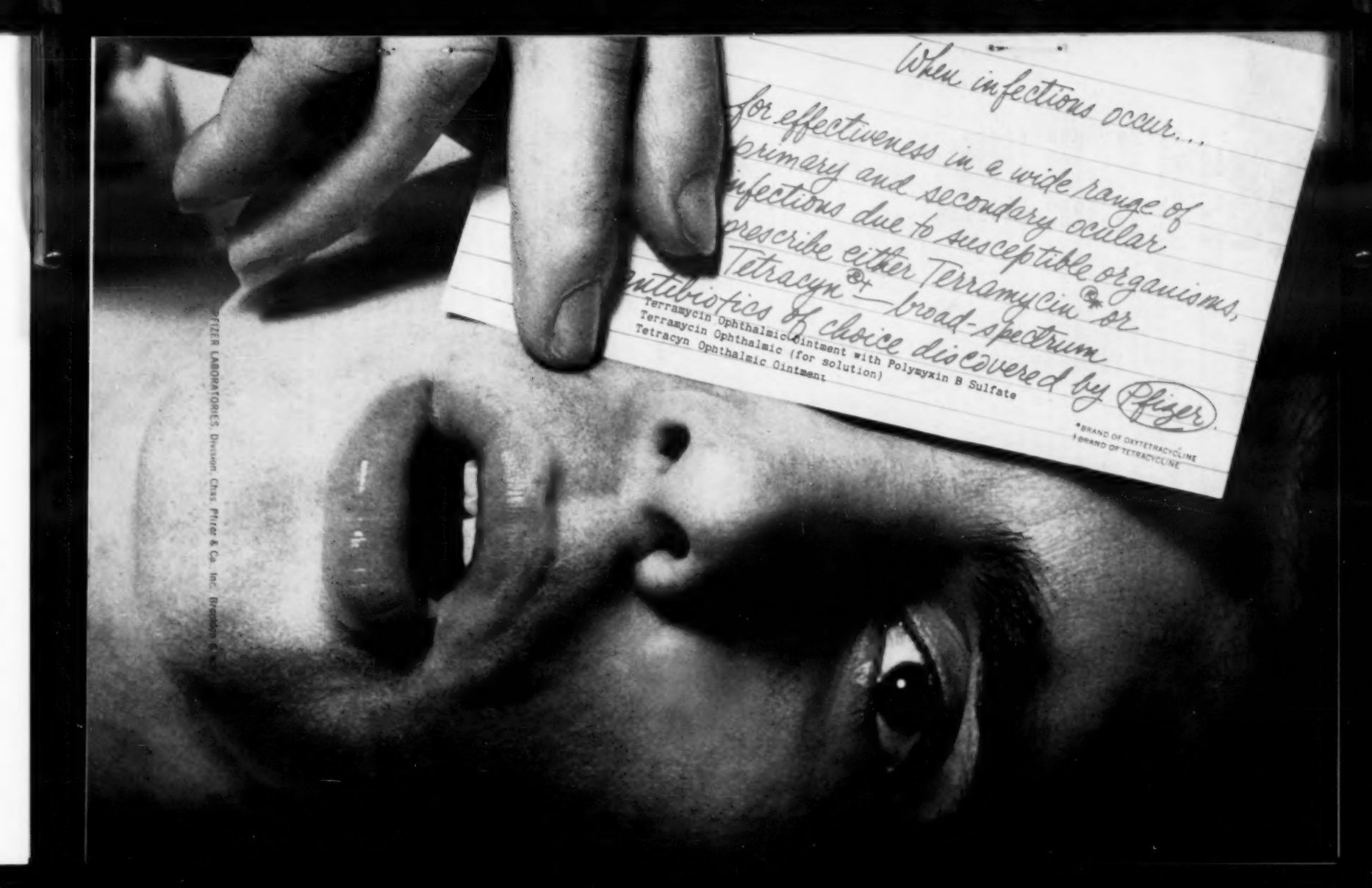
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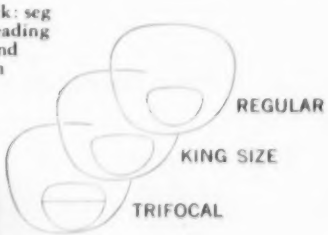

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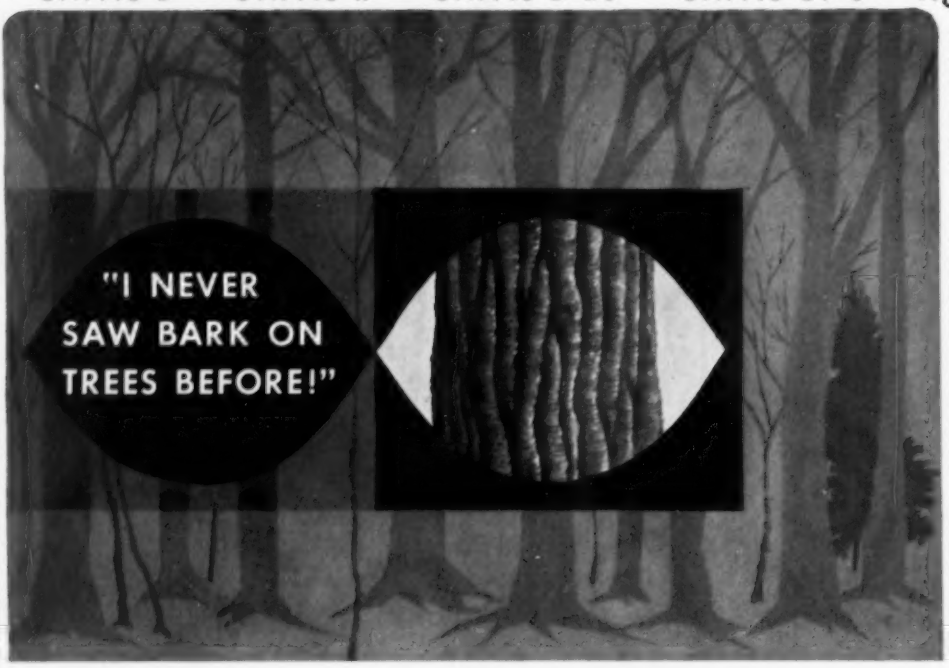

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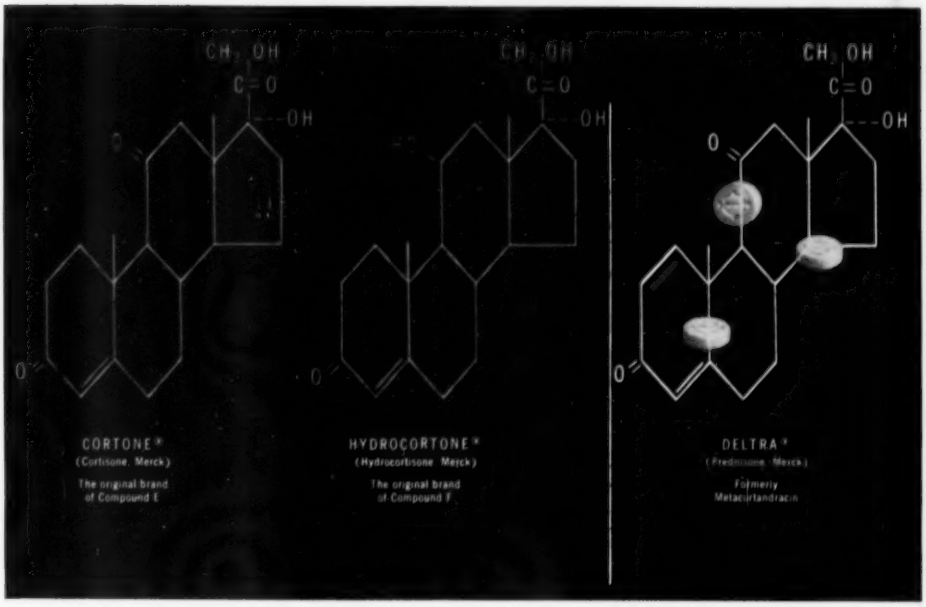

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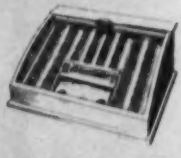

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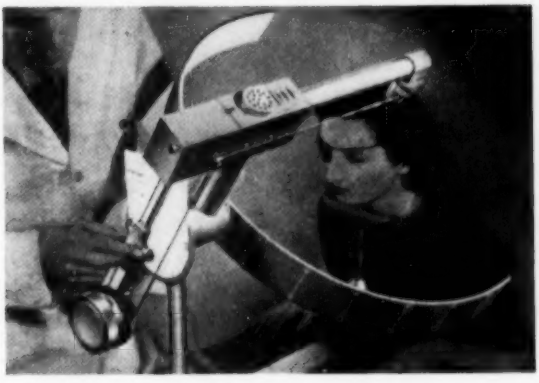

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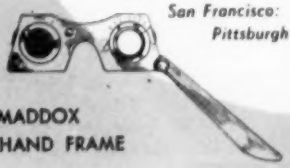

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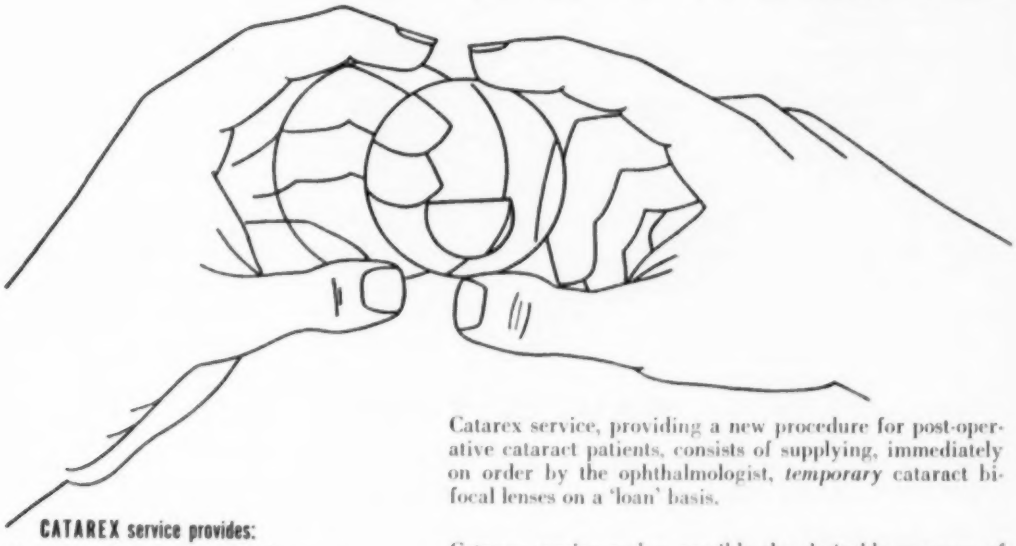

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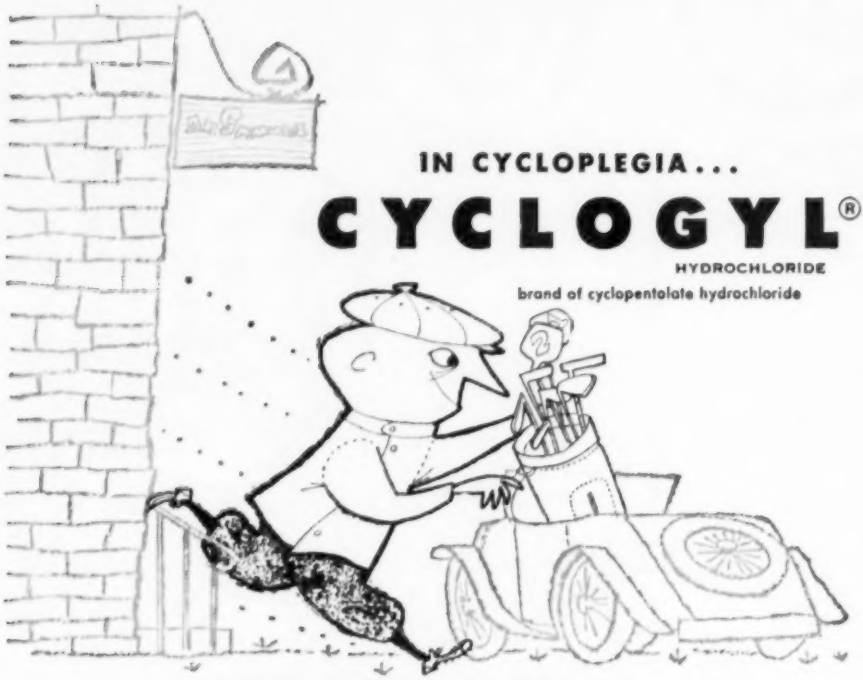

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Fixation of Corneal Grafts

J. W. TUDOR THOMAS, LL.D., D.Sc., M.D., M.S., F.R.C.S., Cardiff, Wales

The subject of corneal transplantation has been of great interest to me for over 30 years, and it is perhaps appropriate that I should address you on a topic associated with that branch of ophthalmological work. I propose in the main to speak on the subject of the fixation of corneal grafts, with particular reference to circumscribed penetrating grafts.

It is now well established that a homoplastic corneal graft will unite readily with other corneal tissue if it is reasonably suited for its reception, and that such a graft can remain transparent. Many things, however, can happen to interfere with the success of a corneal graft, some of which are avoidable; and matters relating to the fixation of the graft are of considerable importance.

The objective in fixation is to maintain the cut surface of the graft in close apposition to its bed, until such time as union has taken place and the mechanical help of fixation devices is no longer required. In addition to this, there are many possible undesirable things which one should try to avoid, such as any unnecessary trauma affecting the graft itself.

The history of corneal transplantation shows that much attention has been given to the matter of fixation, although even to the present day there is no complete unanimity regarding technical procedures.

In the early 19th century attempts at corneal grafting were carried out, often with no special device for fixation except by maintaining lid pressure when the graft was in place. One must, of course, remember the difficulties experienced by these early surgeons, recognizing that ether and chloroform were not introduced as anesthetics until toward the end of the first half of the 19th century, and that the use of cocaine as a local anesthetic was not established until 1884.

In the early part of this century Zirm obtained a clear corneal graft and utilized one overlying suture to maintain it in place. Many surgeons used some form or other of conjunctival flap for that purpose.

Although it is now past history, it might be of interest on this occasion to refer to my experimental work on rabbits, which I started in 1922. At that time there had been very few successful grafts, and there was much uncertainty about the technique to be adopted and the results that might be possible.

In 1930 I read a paper [19] at a meeting of the Ophthalmological Society of the United Kingdom in London, and was able to demonstrate rabbits with clear corneal grafts.

GENERAL PRINCIPLES

As a result of my experimental work, it appeared that there were some general principles which one could follow in order to obtain successful results from perforating keratoplasty in rabbits. They were as follows:

1. Fixation of the graft with crossed sutures
2. Shelving or sloping margins
3. Use of trephines with a difference in size, the smaller trephine being used for removing the graft
4. Prevention of the formation of a fibrinous coagulum by the use of citrated saline solution

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Address of invited foreign guest.

The Sutures.—It often happened in rabbits that the insertion of marginal sutures produced a line of tension across the graft, which caused a suture to cut out rather early, producing a gap between graft and cornea at some part or other. When sutures were arranged to pass over the surface of the graft, it was found that if they took a parallel course, there was a tendency for the suture to come to lie at the margin of the graft sometimes, interfering with union, and it proved much more satisfactory to arrange the overlying sutures so that they crossed at the center of the graft.

Use of Sloping or Shelving Margins.—I still employ this technique as a general rule, although I recognize that many surgeons obtain excellent results by cutting straight through and making no attempt to slope the margins of the graft, and I have used this method myself in some cases. It is, of course, quite easy to cut vertically through the cornea when removing the corneal graft, but it is not so easy to cut vertically through the cornea of the recipient eye, because of the danger of injuring the lens when bold trephining of this kind is carried out. It may therefore happen that in the recipient cornea the margins will tend to slope, particularly in the deeper layers, where removal has to be effected by scissors in parts where the trephine has not completely penetrated. This may lead to some discrepancy between the sharply cut, nonsloping margins of the graft and the bed into which it has to be fitted. If, however, the graft is removed with a technique of trephine and scissors, carried out in a manner similar to that which one would probably have to employ in the recipient eye, the slope of the margins of the two should correspond reasonably well. It is important when removing the graft that there should be as little tearing or bruising of tissues as possible. One might argue that this is likely to be better when a clean cut is made straight through with a sharp trephine. It can, however, be made quite satisfactorily if trephining is carried out to about half the thickness of the cornea and, after an opening is made through into the anterior chamber, the deeper layers are cut with an appropriate and efficient scissors.

The cornea of the rabbit is very thin, and the importance of a shelf is probably greater in the case of the rabbit than in the case of man. On the other hand, when both the graft and its bed are prepared with shelving margins, and the graft secured in place with overlying sutures, a watertight union is provided which is very efficient. At the end of the operation in a straightforward case, it is almost always found that the anterior chamber is partly reestablished, and the iris, with the pupil dilated, is free from the graft. There is usually a slight degree of swelling of the graft in the days following operation, and in rabbits this is often noticeable. If the graft is cut a little smaller than the hole in the recipient cornea, it does allow to some extent for this tendency. Furthermore, when operating on man, one finds the graft tends to fit snugly into its bed, and the tendency toward indenting by overlying sutures is reduced. The watertight union produced by shelving margins perhaps accounts for the fact that in my experience, and in a straightforward case, adhesion of the iris to the graft is an uncommon complication, whereas, I believe, it is more commonly found when the margins are cut without any shelf.

Difference in Size of the Trephines.—This is probably of less importance when one is operating on man than when operating on rabbits. If the difference in size is much, e. g., more than 0.3 mm., one tends to get a narrow furrow around the graft, which is a disadvantage. A slight difference, however, such as 0.1 or 0.2 mm., is, I believe, still an advantage when one is operating on man, particularly so if the margins are cut slightly sloping.

Crossed Sutures.—I described this technique 25 years ago, but I would like to emphasize that when it is carried out properly, the sutures should cross at the center of the graft. Many surgeons have now accepted the principle of two crossed sutures, so arranged that the knot is fixed between

two intracorneal portions of the suture to one side. Two sutures are used, and in order to insert them evenly and accurately, I have been in the habit of marking eight points on the cornea surrounding the graft, indicating where the needle should enter and leave the cornea in each case. Recently I have been marking four dots at 12, 3, 6, and 9 o'clock, instead of eight, which serve equally well as a guide for the correct insertion of the sutures. It is not always easy to make these marks in such a way as to do the minimum of harm to the cornea, and at the same time be easily visible. Materials such as methylrosaniline chloride (gentian violet) jelly can be used, and I am now using a solution of 10% fluorescein, making the marks with the point of a fine knife.

Assuming that two crossed sutures are used, they give eight-point fixation of the graft, and this has proved efficient and reliable when using a graft of about 5 mm. diameter, particularly if the sutures are carefully inserted and their distribution is regular and according to plan. This gives an interval of about 2 mm. at the margin of the graft between adjacent portions of the sutures. If the sutures are placed 1.5 mm. away from the edge of the graft, the intracorneal portion of each suture would be just over 3 mm. in length, so as to arrange for symmetrical distribution of the pattern.

If a large corneal graft is used, 6 or 7 mm. in diameter, it is generally found desirable to utilize interrupted marginal sutures.

Use of Citrated Saline Solution.—This was found to be of great service when operating on rabbits. If no anticoagulant is used, a troublesome clot of fibrin is formed, which not only adds to the difficulties of operation, but tends to produce an adhesion of the iris to the graft. I still use citrated saline when operating on man, and have not found troublesome bleeding resulting from its use, nor have I found it impair the efficient union of the graft.

CORNEAL GRAFT FIXATION

I would now like to trace the tendencies in corneal graft fixation since 1930. At that time Elschnig,[4] who had done much valuable work, was using two overlying sutures to maintain the graft in place, arranged at right angles to each other, and inserted at the limbus.

When surgeons began to take up corneal transplantation, it was evident that many did not like the idea of using sutures to retain a corneal graft in place, and in 1932 Castroviejo[1] advocated the use of conjunctival flaps; but in the course of the next few years he came to the conclusion that sutures were more effective, and introduced a square corneal graft with beveled edges and described a continuous suture which gave eight-point fixation, in the same way as the two crossed sutures.

It was, of course, quite understandable that conjunctival flaps should be tried, but in practice they were unreliable, and one could not have that sense of security with their use which was possible by using sutures.

Katzin described a single suture that gave six-point fixation, but although this method is efficient in securing a smaller graft, the eight-point fixation obtained by two crossed sutures is more efficient for the ordinary 5 mm. graft.

The use of egg membrane between the overlying sutures and the graft was originally suggested by Filatov,[5] and revived and made widely known by Paufique. It is certainly of great advantage, and I have accepted it as part of my normal routine. It diminishes considerably any damage to the graft by the overlying sutures and spreads out the pressure of the suture over a wider area. I have not found any harm resulting by covering the graft with egg membrane in this way, and it allows one to leave sutures in for a longer period without harm.

In my early days, when no egg membrane was used, I generally tried to remove the sutures about the seventh day, recognizing that if they were left in much longer than that, they would produce indenting, and even cutting, of the surface of the graft. This would frequently clear up in a surprising way, but in some cases it produced ulceration

and opacity. Although a graft is united at the end of seven days, the union is insecure, and displacement of the graft margin can easily occur, either at the time of the removal of the sutures or soon afterward, and one cannot regard the union at that stage as being sufficiently secure to withstand possible mishaps. With the use of egg membrane, however, the sutures can be left in for 10 or 12 days, when the union is very much more secure.

Coming to more recent times, we find a tendency to cover the graft with a cap or splint, which is secured to the surrounding cornea. Pittar [8] described a tantalum splint, and in 1952 Worcester [11] published his observations on the use of a corneal splint. The object in view was to preserve the curvature of the graft and to reduce postoperative myopia. It was also intended to prevent the margins of the graft from becoming unduly prominent and to assist in adequate closure of the wound. Worcester first tried a total plastic cap in rabbits, but found it caused considerable opacity and loss of epithelium and evidence of damage to the cornea, in the course of a few days. He concluded that support for the graft must not interfere with adequate respiration of the cornea or with efficient circulation of its fluids. He designed a triangular plastic cap, leaving a portion of the graft surface exposed, and with its use obtained transparent grafts.

Philps [7] also obtained successful corneal grafts with uniform curvatures by the use of a plastic splint, 12 mm. in diameter, which was secured to the cornea and removed after eight days.

Paton [6] is of the opinion that plastic splints tend to interfere with union, and uses a small plastic cap which covers only part of the graft, protecting its central area from the pressure of the sutures.

It is an indication of our progress in corneal grafting that attention is now being given to matters of refinement, which years ago were subsidiary to other, major issues. It is eminently desirable that a corneal graft should have the uniform curvature of a normal cornea and that its surface should be free from irregularities. The use of a plastic splint certainly tends to produce this result, but one must not forget that it is still important for corneal grafts to adhere with as few complications as possible, and without adding to the risks of operation.

In recent years there has been a tendency to use interrupted sutures for corneal grafting. These are necessary when large grafts are used, and many surgeons utilize interrupted sutures as a method of choice for graft fixation generally. This technique has been assisted by the availability of very fine needles, which can be inserted with very little trauma to the graft, and excellent results are reported from operations performed in this way. There are, however, some arguments against the use of interrupted sutures for fixation when overlying sutures can be used. There is some risk of one or two of the sutures cutting out earlier than intended, leaving a marginal gap. There is also some risk of damage to the graft when many sutures are inserted, and the possibility of an uneven pull, causing some distortion of the graft. In expert hands, these difficulties are certainly reduced to a minimum. It has also been found that the removal of sutures is not an easy matter, and Lister cited three cases in each of which removal of interrupted sutures at the 14th day resulted in loss of aqueous. The risks of detaching a portion of the graft when removing the sutures are greater when dealing with interrupted sutures than when dealing with overlying or crossed sutures. Indeed, the suggestion was made that some form of absorbable suture could be used so that one would not have to remove the sutures at all.

The use of plasma and thrombin to produce an immediate fixation of a corneal graft has been advocated, and successfully tried by Tassman,[9] but Dunnington [3] is of the opinion that a fibrinous clot of that kind tends to separate the edges of a wound and that the union so obtained is lacking in normal strength. In my view, it is probably more

desirable to use anticoagulants in the neighborhood of a corneal graft during operation, for reasons which I have explained.

Accuracy of apposition of the margin of the graft with its bed is, of course, very necessary. Although this can be obtained by the use of interrupted sutures, or by the use of a plastic splint, it can also be obtained by the use of shelving margins, provided the technique of preparing these margins is carefully carried out. It is important to choose very carefully a scissors that will cut neatly and decisively as required. There are several efficient scissors of this type which are available.

It is still my practice to prepare the graft with shelving margins, to utilize a slightly smaller trephine for outlining the graft, and to secure the graft in place with two overlying sutures. The sutures are left in place for 10 or 12 days, and this is made possible by the use of egg membrane. The slight disadvantage attached to egg membrane is that it is opaque, and at the end of operation one cannot actually see the graft and its position, although any displacement should be evident and should be at once rectified.

With this technique, and in a favorable eye, my experience has been that there is little tendency to produce the undesirable complications that plastic splints are intended to overcome, namely, prominence of the graft margins and postoperative myopia. There is also the advantage that one can leave the sutures in place and undisturbed until 10 or 12 days after operation, without risk of possible ill effects that might follow if complete covering of the cornea were carried out by a plastic splint for so long a time.

Efficient fixation of a corneal graft is of fundamental importance, and it is evident that this can be brought about in several ways. Time will show whether one way is safer and more secure than another, and by free interchange of our experiences we will be able to assess the merits of variations in technique.

I have watched with admiration the progress in keratoplasty which has been going on in the States for many years past, and which has been of such benefit to us all.

It is a tremendous advantage which the world now enjoys that we can so freely exchange information in this branch of ophthalmology for our mutual benefit.

16 Cathedral Rd.

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Etiology and Therapy of Retinal Vascular Occlusions

BANKS ANDERSON, M.D.
and
WILLIAM VALLOTTON, M.D., Durham, N. C.

Retinal diseases fall into two large groups: (1) those which develop as a result of circulatory disturbances and find expression in retinopathies more or less intimately associated with the retinal vessels, and (2) those extravascular lesions which are often hereditary and almost always degenerative in character. The latter have no place in this discussion. Retinopathies resulting from circulatory disturbances may be entirely intravascular in type or may be accompanied by edema, exudates, or hemorrhages as extravascular components of the same underlying process. In such cases the extravascular component usually is a quantitative expression, an overflow as it were, of a process no longer controlled by or contained within the retinal vessels. The increase in permeability which permits this pathologic movement of fluid or formed elements into extravascular space may, in turn, be due not only to structural or functional modifications of the lumen and walls of the vessels but also to qualitative and quantitative changes of the blood. Thus it is that the structure and function of the retina may be affected by disease of the blood-forming organs; by abnormalities of the heart and great vessels resulting in changes in the character, volume, and pressure of the blood stream; by disease of the cranial blood vessels, and, more immediately and profoundly, by changes in the retinal vessels, which occur either independently or in a well-recognized and established association with any of the above conditions. We are here concerned with the early recognition of such diseases and with a survey of the therapeutic resources which may be utilized in preventing retinal damage and in promoting the regression of those lesions which may have already developed. The changes in the retinal vessels which are of most concern are the occlusions of the central trunks. The hemorrhages, exudates, proliferations, and neovascularizations which precede or follow such a catastrophe are of relative minor importance.

Obstruction of the retinal vein most frequently results in visual loss—occlusion of the retinal artery almost always does so. Occasionally, although the appearance of the fundus indicates severe circulatory embarrassment, function is preserved or restored. Those few instances in which vision has been salvaged, stray beams of light in an otherwise dark and hopeless picture, have prompted us to examine our own experiences in this desperate situation and to review the investigations of others. The question posed is this: In retinal arterial and venous occlusions, is the preservation or restoration of vision entirely fortuitous, or are there in such cases clear indications of effective therapy? The cases selected for this study represent our personal experience during the last decade. In arterial occlusion the criteria for inclusion in the study consisted of the appearance or persistence of the characteristic cherry-red spot. Under such a standard a certain number of cases which later resolved into branch ob-

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From the Division of Ophthalmology, Duke University School of Medicine and Duke Hospital

struction have been included. The 56 cases studied probably represent the great majority of the arterial obstructions examined in this period. On the venous side, the standard for inclusion in the study has been clear-cut obstruction of a branch by a rigid artery or the characteristic overripe smashed-tomato appearance of the central venous trunk occlusion. Probably no high percentage of branch venous occlusions has been recovered from the files, as these lesions have been seldom coded.

After careful evaluation of all the available evidence, we have felt that in many instances one could make a reasonable assumption of a correlation between the retinal lesion and some other factor in the history or examination which would permit the division of these cases into etiological groups. For instance, where the diastolic blood pressure was above 100 mm. and the patient over 50, classification has been entered under the hypertensive-arteriosclerosis column. Likewise, when there was a clear-cut history of rheumatic fever with other residual evidences of such an infection, we have felt there most probably was a causal relationship between this past disease and the occlusion. When nothing in the history or examination of the patient was indicative of a systemic disease as a contributory factor or when such evidence was weak, the case has been entered in the unknown column. Under such a plan, our material breaks into the following distribution, arranged in part on a presumed etiological basis, but with the intraocular and extraocular components overlapping in anatomical sequence (Table 1).

Table 1 demonstrates clearly (1) that the great majority of occlusions are associated with arteriosclerosis and hypertension, and (2) that the same diseases listed as presumably responsible for, or at least associated with, the occlusion are implicated in both arterial and venous occlusions. Since there is likewise an overlap in the therapeutic procedure, we have chosen to consider insofar as possible the two types of occlusion as a single problem.

In all these obstructions, from whatever cause, the immediate therapeutic aim is that of increasing the caliber of the vessel which has become constricted beyond the point of satisfactory functioning. For this purpose we have available therapeutic agents which are presumed to relax vasospasm and anti-

TABLE 1.—*Etiological Classification of Retinal Vascular Occlusions*

	Arterial		Venous	
	Partial	Total	Partial	Total
Infectious vasculitis				
Syphilis	..	1	..	1
Tuberculosis	..	..	2	..
Postrheumatic fever	2	3	..	..
Hepatitis	..	1	..	..
Unspecified or unknown	2	2	2	3
Trauma				
Pressure	..	2	..	..
Hydrogen peroxide in antrum	..	2	..	..
Hypertensive arteriosclerosis	6	11	16	18
Diabetes	..	..	1	1
Diseases of blood				
Polycythemia	..	2	1	..
Leukemia	..	..	..	1
Collagen disease				
Lupus erythematosus	..	1	..	..
von Hippel-Lindau angiomatosis	..	..	1	..
Unknown	4	6	6	8
Cavernous sinus thrombosis	..	1	..	1
Arteriovenous fistula	..	2	1	..
Temporal arteritis	..	5	..	..
Carotid artery occlusion	1	1	..	..

coagulants which reduce the chances of further thrombosis. Our problem lies in bringing these agents into effective use in properly selected cases. Our difficulty is in differentiating such cases from those in which the occlusion is complete, with organic irreversible closure. Such a differentiation is clinically impossible. We feel that all such cases when seen early—arterial, within the first week; venous, within the first month—should be treated expectantly. In such an unselected group, one cannot expect a high percentage of success. There may be, as has been demonstrated by Verhoeff, Klein, and others, anatomicopathologic relationships which reduce or neutralize our therapeutic efforts. For instance, a sclerotic retinal artery may well retain sufficient lumen to meet the needs of a given sector of the retina but, by the rigidity of its wall and the constrictive effect of the sclerosing process, may block the return flow at an A-V crossing. This process, in our present knowledge, is irreversible. Our therapeutic concept in such cases must be oriented to prevention of the organic change in the arterial wall, since we cannot change the anatomical relationship of artery to vein. If, on the other hand, in another case we accept the hypothesis that the constrictive effect is in part the result of vasospasm in hypertension, or is due to the inflammatory thickening of the vessel wall or to the development of a thrombus on a damaged endothelium, therapy may be effective. In such cases our hope of successful therapy lies in reduction of the blood pressure and release of the vasospasm, or in control of the inflammation within the vessel wall, or in the alteration of the character and pressure of the contained blood. For, while we cannot conceive of any emergency therapy which would be effective in purely sclerotic thickening of an arterial wall or in reducing the purely mechanical pressure of such a vessel upon an underlying vein, we can consider the possibility of relieving the spasm of a retinal artery induced by an embolus, and possibly in assisting in the migration of such an embolus to a less important area of the vascular tree. We do have available anti-inflammatory agents which may be capable of reducing the inflammatory thickening of vessel walls. There are agents for reducing the intraocular pressure, and by this means possibly raising the relative level of the pressure of the entering blood. Finally, anticoagulant therapy has been proved effective in the control of thromboses and thrombophlebitis in other areas. On theoretical grounds, we have every reason to expect arterial and venous occlusions, other than those due to arteriosclerosis, to be favorably influenced by such therapy.

TREATMENT

Vasodilators in Arterial Occlusions

In the 10-year period during which this series of occlusions has accumulated, theoretical considerations in treatment have not changed. Newer and more effective drugs, as they became available, have replaced their older and less effective counterparts. Currently, our procedure follows somewhat the following pattern: Treatment is recommended for all arterial occlusions seen within the first week, and for all venous occlusions seen within the first month, after the onset. In spite of the preceding remarks regarding the hopelessness of cases due to arteriosclerosis, we feel that, unless the associated hypertension is so severe and the apprehension so great as to make any manipulation physically and psychologically hazardous, the patient should be given the advantage of even this slight chance of preserving visual function. Even in these cases we cannot be sure that the process is irreversible. Moreover, anticoagulant and antispasmodic therapy in such cases may prevent other, and more serious, circulatory failure, which may be imminent in more vital organs. Finally, though vision may not be restored, a therapeutic plan properly instituted will at least have the psychological advantage of cushioning the blow—conditioning the patient to the disability.

As soon as possible after the preliminary examination a retrobulbar injection is done. This may be repeated in four to eight hours. Currently, we are using 2% procaine hydro-

chloride combined with 250 units of hyaluronidase. Two cubic centimeters is injected. Since the pupil can be observed to dilate with retrobulbar procaine, it is assumed that the vasomotor fibers to the retinal vessels (if such exist) have also been acted upon by the drug. We are also using tolazoline (Priscoline), 10 to 25 mg., for the initial retrobulbar injection, or for the subsequent retrobulbar injection if more thorough examination indicates that the patient has no evidence of coronary disease, gastritis, or other contraindicating disease. One possible disadvantage of tolazoline is the fact that, acting as it does on the end-organ of the sympathetic nerve fiber within the vessel wall, absorption into the blood stream results in a general lowering of the blood pressure. This neutralizes the effect of the hyaluronidase injected, with the idea that lowering the intraocular pressure might create a favorable situation for the vis a tergo effect of the blood column in moving an embolus or distending a vessel. We have seen acetylcholine used in one case as a retrobulbar block. This patient, who had a complete occlusion, with the formation of a cherry-red spot, recovered 20/20 vision. This drug, for reasons to appear later, is not now recommended.

The next development in our routine calls for a block of the vasoconstrictor fibers at the stellate ganglion. In our hospital this is done by the anesthesiologist. Few ophthalmologists would feel inclined to attempt such an approach, although we are told that this procedure is neither as difficult nor as hazardous as a spinal tap. If competent personnel is available, such a procedure should be used to augment, or as an alternate method of releasing, whatever vasoconstrictor effect there may be present in the retinal vascular tree.

Anticoagulant Therapy in Arterial and Venous Occlusions

In arterial occlusion, as soon as possible after hospitalization, anticoagulation therapy is instituted. In this respect the treatment of the arterial and of the venous occlusions coincides. Heparin sodium (Depo-heparin sodium) in a 300 to 400 mg. dose according to weight is immediately given subcutaneously, followed by a slightly smaller amount in 24 hours to maintain a clotting time of approximately 20 to 30 minutes. A third injection may be given the third day if bishydroxycoumarin (Dicumarol) or ethyl biscoumacetate (Troxmexan), which was also given by mouth on admission, has not become effective. Bishydroxycoumarin is given in 300 mg. amounts on the first day, 200 mg. doses the second day, and 50 to 100 mg. doses thereafter, in order to keep the prothrombin time at a level of 20% to 30%. During hospitalization ethyl biscoumacetate may be used in preference to bishydroxycoumarin. This is a more rapidly acting drug. The initial dose is 1800 mg., followed on the second day by 600 or 800 mg., and 150 mg. daily thereafter. Bishydroxycoumarin in daily amounts of 75 to 100 mg. for the average ambulant, nonhospitalized patient is preferred. This amount will usually keep the prothrombin time at a level of 20% to 30%.

In the past we have also used nicotinic acid in flushing doses, although recent evidence tends to invalidate this as an effective dilator of cerebral and ocular blood vessels.

We have felt it worth while to continue anticoagulant therapy for 30 to 60 days in most cases. While this would scarcely be worth while in consideration of the vascular occlusion alone, it is well to remember that the ocular disease in man is only a surface manifestation of a more profound disturbance. One of our untreated patients with arterial occlusion, 24 years of age, died suddenly within a few weeks after the arterial occlusion. Another man, mentioned above as recovering normal vision after arterial occlusion treated with acetylcholine, died suddenly with coronary occlusion some four years after the ocular difficulty.

In two of the later cases of temporal arteritis corticotropin and cortisone were used unsuccessfully in an effort to reduce the inflammatory thickening of the arterial

wall. Phenylbutazone (Butazolidin), a nonhormonal anti-inflammatory agent, might also be considered in this connection.

RESULTS

As anticipated, our results have been disappointing. The results in arterial occlusion are shown in Table 2. Those in venous occlusion are shown in Table 3.

COMPARISON WITH RESULTS REPORTED IN OTHER STUDIES

Although, on the basis of the therapeutic results which are reported here, one cannot develop any great enthusiasm for the treatment of retinal vascular occlusion, the picture is even more hopeless in studies reported before the introduction of the vasodilator and anticoagulant drugs. Foster Moore,[1] in his 1924 monograph on retinal venous thrombosis, followed 19 cases of central occlusion, the majority of them from 5 to 10 years. He reported that "in no instance where it [vision] is so good as 6/60 six months after thrombosis . . . one can say then of these cases that it is quite unusual for any useful sight ultimately to be left." In 1942 Odqvist, as reported by Brændstrup,[2] found that of 36 cases of untreated central vein thromboses vision had returned to normal in 5; 11 patients were able to read; 12 were able to orient themselves with the previously affected eye, and 8 had become blind. Brændstrup remarked that "the author [Odqvist] finds the function prognosis better than is usually reckoned with." Brændstrup studied 131 cases of central vein thrombosis from two Danish hospitals. His patients had not had anticoagulant therapy. In this series he found that as the final visual result, 45% had an

TABLE 2.—*Arterial Occlusions, Branch and Trunk*

	Treated	Improved	Untreated	Improved
Infectious vasculitis				
Syphilis	..	..	1	0
Postrheumatic fever	3	1	2	0
Hepatitis	1	0	..	..
Unspecified or unknown	1	1	4	0
Trauma				
Pressure	..	..	2	0
Hydrogen peroxide in antrum	..	..	2	0
Hypertensive arteriosclerosis	5	1	13	2
Diseases of blood				
Polycythemia	2	1	..	..
Collagen disease				
Lupus erythematosus	..	..	1	0
Unknown	4	2	6	0
Cavernous sinus thrombosis	..	..	1	0
Arteriovenous fistula	1	0	1	0
Temporal arteritis	2	1	3	0
Carotid artery occlusion	1	0	1	0

TABLE 3.—*Venous Occlusion, Branch and Trunk*

	Treated	Improved	Untreated	Improved
Infectious vasculitis				
Syphilis	..	..	1	0
Tuberculosis	..	..	2	0
Unspecified or unknown	3	3	2	0
Hypertensive arteriosclerosis	5	2	29	4
Diabetes	..	..	2	0
Diseases of blood				
Polycythemia	1	0	..	..
Leukemia	..	..	1	0
von Hippel-Lindau angiomatosis	..	..	1	0
Unknown	8	4	6	2
Cavernous sinus thrombosis	1	0	..	..
Arteriovenous fistula	..	..	1	0

acuity above, and 55% an acuity below, 6/60. He found that the youngest patients obtained the best acuity. Finally, Duff, Falls, and Linman,[3] after a most searching analysis of the world literature and an evaluation of their own material, reported that in a control series of 79 untreated cases of complete central vein occlusion the condition of 49 grew worse, while secondary glaucoma developed in 43%; only 15% showed improvement, whereas the condition of 35% remained unchanged. According to these authors, the collected experience with anticoagulant therapy after thrombosis of a tributary or central retinal vein indicates that in 58% the prognosis for visual improvement is favorably influenced and the frequency of secondary glaucoma encouragingly lowered.

Our experience with vasodilators and anticoagulants, on the basis of our small number of cases, leaves us with an open mind. In six patients, three of whom were sufficiently young that arteriosclerosis presumably could be ruled out, we were unable to demonstrate any change in the caliber of the retinal vessels by either observation or photographs taken immediately before and after both cervical block and retrobulbar injection. Furthermore, we have never been completely convinced that we could observe a significant change in the caliber of the vessels observed in the operating room before and after retrobulbar injection. This raises the question of the degree, if any, of the vasomotor control over the retinal artery. Wagener [4] reported in 1931 his observations of dilatation of both veins and arteries after resection of the inferior cervical and first and second thoracic ganglia. In these cases is the dilatation due to the resection per se or to the resulting lowered blood pressure? However, Michaelson [5] reported a 41-year-old woman with a branch block which had been present for six days. Fifteen minutes after the retrobulbar injection of 0.1 gm. of acetylcholine "the white opacity of the retina was noted to be markedly diminished and to be confined almost entirely to the area between the affected artery and macular vein." During the next few days the opacity disappeared completely. Michaelson observed that in this case there was no notable change in the diameter of the other retinal vessels. He stated that he had noted a similar lack of general vascular response to this drug in two other patients.

Payne and Reed [6] traced 77 cases of retinal artery occlusion in the Royal Eye Hospital. In only one was any improvement in vision noted. Twenty-two of these patients were treated by retrobulbar injections of acetylcholine, 0.05 gm. Only one, a 77-year-old patient treated within 12 hours after occlusion, showed improvement from light perception to 6/12.

More encouraging is the report of Weerekoon,[7] who collected from the literature a case reported by Thompson in which the diagnosis was made 25 minutes after onset and function restored to normal by vasodilator drugs, five cases of Thiel's treated by retrobulbar tolazoline (Priscol), with recovery in one case, and three cases from the Western Ophthalmic Hospital, in two of which central visual acuity was recovered, though the field defects were still present.

Coston [8] reported three cases of arterial occlusion improved by stellate ganglion block. Coston made the point that ganglion block was preferred because the vasodilator fibers lying within the nerve were less accessible to the blocking agent in retrobulbar injection. Guillette and Mayer [9] also reported the dramatic recovery of a 37-year-old woman who was so fortunate as to have treatment begun 20 minutes after the occlusion.

SUMMARY AND CONCLUSIONS

An attempt has been made to differentiate etiologically 120 cases of retinal arterial and venous occlusion. Arteriosclerosis and hypertension could be causally related to one-third of the arterial occlusions and to one-half of the venous occlusions. In this group there was no evidence of therapeutic effect on the arterial occlusions, but, from the limited number of cases available for comparison, therapy might be beneficial in the venous obstructions. Therapy seemed to be most effective in the infectious or postinfectious

group in both the arterial and the venous obstructions. In the unknown group, on the arterial side, two of four occlusions improved under therapy, while no improvement was noted in six untreated cases. On the venous side in this group the results were equivocal. The failure of corticotropin in the two cases of temporal arteritis was disappointing. The cases differentiated under other etiological groups were not of sufficient number to indicate any trend in therapeutic effect. Other studies which have preceded this report are in general accord with our conclusion that anticoagulant therapy is justified in both arterial and venous retinal occlusions; that the results in the hypertensive-arteriosclerosis group will be poor, but that in those occlusions associated with infectious processes therapy adds appreciably to the possibility of restoration of function.

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Some Instructive Manifestations of Chiasmal Disease

One Case of Pituitary Adenoma, One of Optochiasmal Neuritis (Arachnoiditis), and One of Chiasmal Glioma

JOHN P. WENDLAND, M.D., Minneapolis

Patients with lesions in the neighborhood of the chiasm usually complain first of visual symptoms. The responsibility for diagnosis of these lesions therefore rests primarily upon the ophthalmologist. When one considers that almost one in four brain tumors [1] lies in the chiasmal region and that chiasmal lesions often threaten not only the vision in both eyes but life itself, it is apparent that the responsibility is great.

CHARACTERISTICS OF CHIASMAL LESIONS

All of us are aware that a bitemporal hemianopsia is a classic finding in a chiasmal lesion. However, we often forget that this hemianopsia is frequently not present in pure form. It is often masked by overlying variations, and occasionally it is not present at all. There are, nevertheless, certain field findings which are of localizing value in chiasmal lesions. These are as follows:

1. A bitemporal hemianopsia with "steps" along the vertical meridian in either one or both eyes, even though extremely asymmetrical, is absolutely diagnostic of a chiasmal lesion. If the defects are fairly equal in the two eyes and more marked in the upper fields, the lesion is midline and most likely beneath the chiasm. Bitemporal hemianopic scotomas also indicate a lesion of the chiasm. The lesion is not always at the posterior portion of the chiasm.[2]

2. A central or centrocecal scotoma combined with a temporal field defect in the opposite eye is usually due to a lesion of the chiasm at the junction of the optic nerve and the chiasm. A "junction" scotoma (a temporal hemianopic scotoma in one eye) combined with a temporal defect in the opposite eye is diagnostic of a chiasmal lesion. The lesion is on the side of the eye with the scotoma.

3. Blindness in one eye with temporal hemianopsia in the other eye usually indicates a chiasmal lesion. The lesion is on the side of the eye which is blind.

4. A homonymous hemianopsia which is very incongruous may be found in early lesions pressing anywhere on the side of the chiasm from the lateral side of its junction with the optic nerve on back to the tracts. However, only in the case of a tract lesion adjacent to the chiasm may a homonymous hemianopsia become very severe without losing its pure homonymous characteristics.

5. A binasal hemianopsia, when caused by a single lesion, indicates pressure on the lateral sides of the optic nerves near their chiasmal junction.

One can see from a listing of these "guideposts" that the field defects of the crossroads of the visual pathways may be diverse. When one also adds to this the fact that the ophthalmoscopic examination may be negative, one is not surprised that early diagnosis may prove difficult. A great deal can be learned

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From the Department of Ophthalmology, University of Minnesota Medical School.

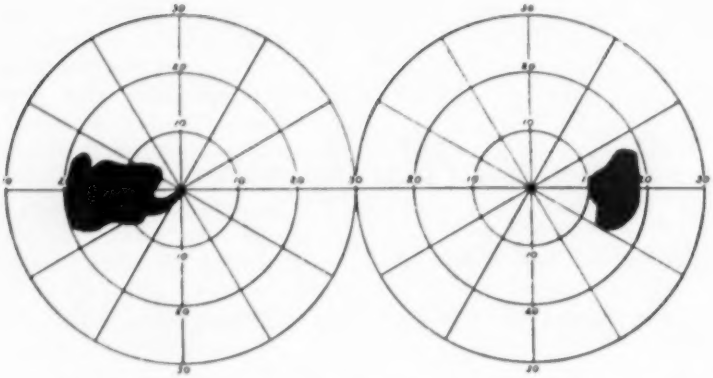

Fig. 1 (Case 1; test object: 1.5 mm/1000 mm.).—Fields obtained by ophthalmologist shortly after patient complained of poor vision in left eye. V. A.: R. E. 20/20; L. E. 20/30.

by the study of individual cases, and it is the purpose of this paper to present a few cases, each of which I feel emphasizes certain features of chiasmal disease which may be easily overlooked.

REPORT OF CASES

Case 1.—This patient was a 52-year-old man who consulted an ophthalmologist shortly after onset of poor vision in the left eye. Fields ("central") were as indicated in Figure 1. Visual acuity was R. E. 20/20, L. E. 20/30. The fundi were normal, and the ophthalmologist made a diagnosis of retrobulbar neuritis. The patient was treated with vasodilators and vitamins.

One year later the patient complained of failure of vision in the right eye also. Fields were as indicated in Figure 2. One may note the presence of bilateral cecocentral scotoma, very suggestive of a toxic amblyopia. Because of the nature of the early field findings, the ophthalmologist became convinced the patient had a "retrobulbar neuritis," and consequently subsequent typical field changes failed to dislodge him from this opinion as soon as they should have. Three months later the fields were as shown in Figure 3. Visual acuity was R. E. 20/400, L. E. 20/30. These fields have classic chiasmal characteristics (temporal hemianopsia in one eye and central scotoma in the other). Two months later the fields were as in Figure 4. The discs showed at this time "slight pallor" with cupping. Intraocular tension was normal. Improvement in the fields encouraged the ophthalmologist to believe that his original diagnosis was correct. The fields, however, were still characteristic of a chiasmal lesion. Seven months later, peripheral fields were done for the first time and skull films were taken which showed enlargement of the sella

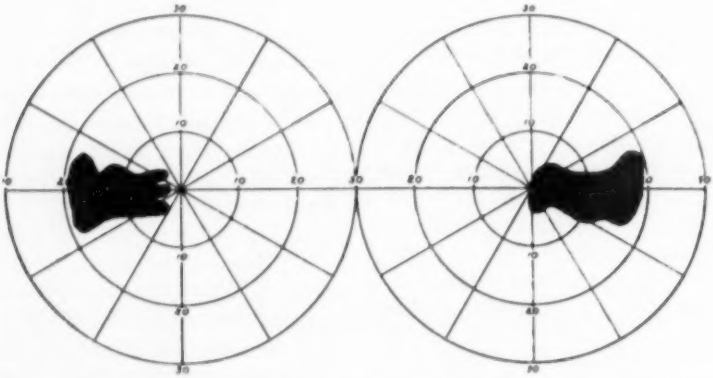

Fig. 2 (Case 1; test object: 1.5 mm/1000 mm.).—Fields obtained one year after fields shown in Figure 1. V. A.: R. E. 20/100; L. E. 20/30.

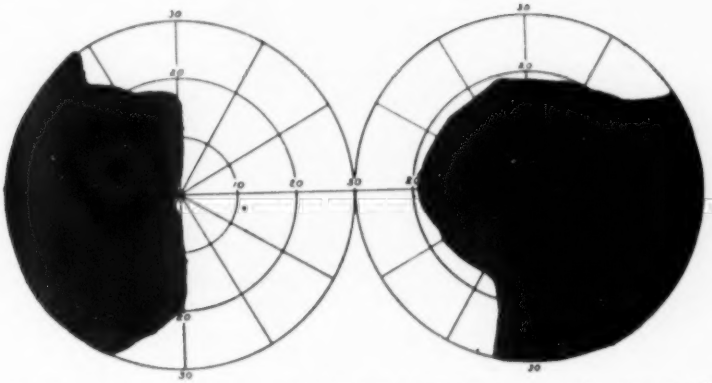

Fig. 3 (Case 1; test object: 1.5 mm/1000 mm.).—Fields three months after the fields in Figure 2, showing unmistakable evidence of a chiasmal lesion. V. A.: R. E. 20/400; L. E. 20/30.

turcica and thinning and posterior displacement of the posterior clinoid processes. Surgery revealed a chromophobe adenoma. Two and one-half years later there was very little change in the fields or visual acuity.

Comment

This case illustrates how closely a tumor affecting the chiasm may simulate a retrobulbar neuritis in its early stages. Various authors * have remarked upon a central scotoma in chiasmal lesions mimicking a retrobulbar neuritis. Simply being aware of this possibility would have aided the ophthalmologist in making the diagnosis earlier. In retrospect (and remembering that it is always easier to be analytical after the diagnosis is made), the scotomas shown in Figure 2 were probably "junction scotomas" and were thus in reality bitemporal hemianopic central scotomas caused by pressure of the tumor on the medial sides of the optic nerves at their junction with the chiasm. It would also have been of considerable help had peripheral fields been taken earlier, as they might have shown superimposed bitemporal defects. Even more important, possibly, would have been the use of a slightly smaller test object on "central fields." This might have revealed some bitemporal characteristics. Chamlin * has emphasized the "intermediate field area" in the early detection of

* References 3 through 7.

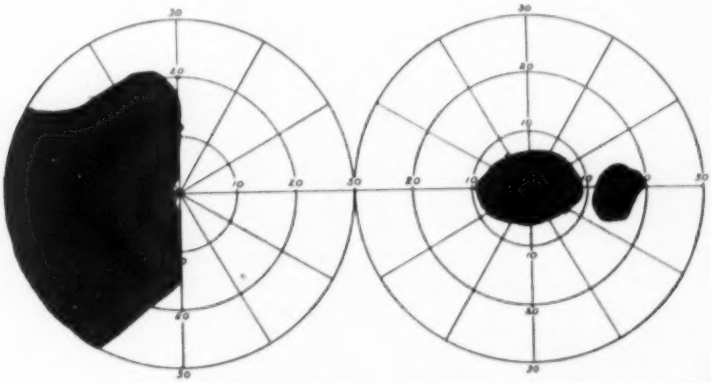

Fig. 4 (Case 1; test object: 1.5 mm/1000 mm.).—Fields two months after the fields in Figure 3, showing improvement in the right eye. This resulted in unfortunate, unwarranted reassurance that the condition was improving.

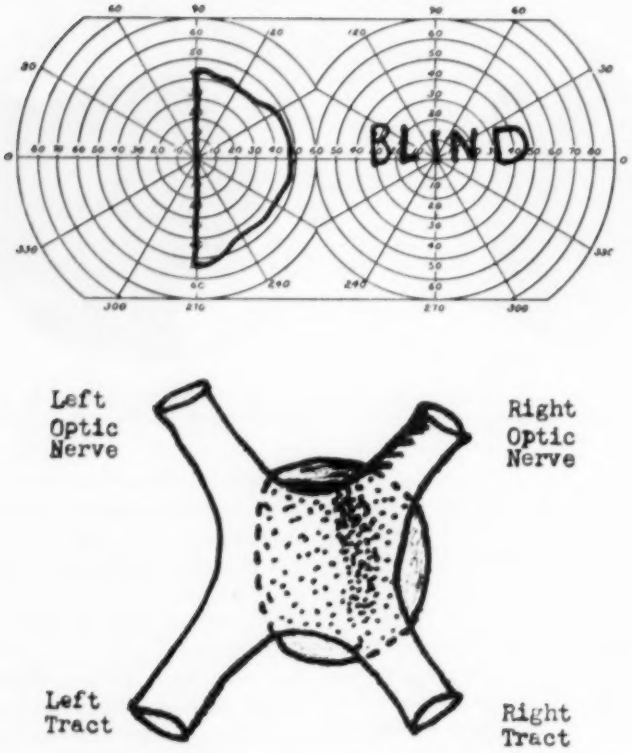

Fig. 5 (Case 1; test object: 3 mm/330 mm.).—Fields just before surgery and position of tumor found at operation seven months after the fields in Figure 4. V. A.: R. E. blind; L. E. 20/80.

chiasmal lesions. Skull films performed much sooner would probably have revealed changes in the sella. It is not necessary, however, to have positive x-ray findings to make a diagnosis of a chiasmal lesion. Walsh,[9] in 50 cases of intrasellar tumors, found x-ray changes in only 65% but observed field changes in 88%. One can thus err by placing too much reliance on negative x-ray findings.

This patient illustrates how variation in the field findings from time to time may also be found in tumors, apparently because of change in position of the tumor or because of recurring hemorrhages in and about the tumor. The over-all course of the field changes is gradually downhill, however, and this trend may be of some help in differentiating an inflammatory process from a neoplastic one.

Case 2.—This patient was a 13-year-old boy who complained of poor vision of six months' duration. He was uncertain as to how rapidly his vision had failed. Visual acuity was R. E. 7/200, L. E. 7/200. The discs were slightly pale. The fields are shown in Figure 6. One may note the bitemporal depression in the internal isopters. The child lived in a rural community and had no history of exposure to any substances which could be considered as toxic chemicals. Except for slight obesity and the field findings, general and neurological examinations were negative. Laboratory studies were normal. An x-ray showed questionable suprasellar calcification. On the basis of the field findings, slight obesity, and questionable x-ray findings, a preoperative diagnosis of craniopharyngioma was considered most likely. However, the possibility of an inflammatory or unknown toxic process involving the chiasm and optic nerves was also considered. At operation nothing was found that could adequately account for the visual loss. There were a few arachnoid "adhesions" and cystic collections of fluid around the chiasm, but the

neurosurgeon stated that in his opinion they could not account for the visual loss. For a time after surgery the patient's vision showed no change, but gradually the peripheral fields became severely involved, until five years later he was for practical purposes blind in both eyes. The optic discs became chalky-white with sharp margins and narrowed retinal vessels. Repeated skull x-rays showed no change from the original films.

Comment

It is interesting to make a comparison of Case 1 and Case 2. Although both cases presented central or cecocentral scotomas, one patient had a tumor; the other did not. It is thus apparent that the presence of a scotoma does not help in giving the diagnosis as to the cause of the lesion. However, it is to be noted that the scotoma, when first noted in the first case, did not depress visual acuity appreciably. The scotomas in the second case when first noted were responsible for pronounced reduction in visual acuity, the vision being only 7/200 in each eye; yet the intermediate and peripheral fields were only slightly affected. I think that as a general rule an inflammatory or a toxic process is more apt to give pronounced loss in visual acuity, with relatively little involvement of the remainder of the field, than is a tumor. This is to be taken as a general rule only, however, as tumors of the chiasm and posterior portion of the optic nerves may on occasion give very dense central scotomas with relatively little involvement of the peripheral fields. Also in favor of tumor, as against an inflammatory or a toxic process, is the presence of bitemporal hemianopic scotomas, as opposed to central scotomas, which freely include the fixation point and which may be of either tumor or inflammatory origin.

Case 2 falls in the category of cases presently labeled as optochiasmic arachnoiditis. This condition was first described independently in 1929 by Cushing and Eisenhardt [10] and by Balado and Satanowsky.[11] Since that time numerous articles have appeared in the literature, and Vail [12] in 1938 gave an excellent description of the clinical features of the disease. Important in the diagnosis is the fairly abrupt onset of a central scotoma in one or both eyes, combined, as time goes by, with more peripheral temporal field defects. The victims of the disease are usually young people, but in one series reported [13] the largest number of cases fell between 41 and 50 years of age. Numerous authors † have emphasized the presence of a mixed type of optic atrophy. The disc is pale, but its margins are sharp; the lamina cribrosa is visible, and the vessels are narrow. Because of variations in the discs, however, this sign is but suggestive. There are, indeed, no pathognomonic signs of the disease. Pneumoencephalography ‡ may be of considerable help in differentiating tumor and the condition

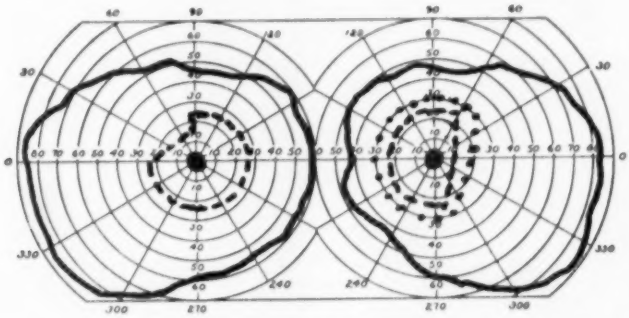

Fig. 6 (Case 2; test objects: .3 mm/330 mm.; 1 mm/1000 mm.; .3 mm/1000 mm.).—Fields obtained just before surgery.

† References 12, 14, and 18.

‡ References 19 and 20.

called optochiasmatic arachnoiditis. The natural history of the disease is variable, with remissions, but in general the outlook is poor as far as vision is concerned. The etiology of the disease is in the majority of instances obscure. Syphilis is one known cause. The very name of the disease suggests that the cause of the loss of vision is the arachnoiditis present, and emphasis has been placed upon the surgical freeing of the adhesions as a means of improving vision. Critical appraisal of the operative results indicate that surgery actually often is of no help. Feld and Auvert,[21] in a series of 148 cases, found only 28% benefited by surgery.

The patient in Case 2, although presenting the "typical" findings of optochiasmic "arachnoiditis," obviously had no visual loss from the "arachnoiditis." The pathology responsible for his visual loss undoubtedly lay within the optic nerves and chiasm itself. Other authors § have suggested that the causative pathology lies in the visual pathways themselves. In many cases any arachnoiditis present would appear to be a process concomitant with the process going on in the visual pathways themselves, and not the cause of the visual loss. The neurosurgeon must have the courage to state that he found nothing really to account for the visual loss. The etiology of the process present in the optic nerve and chiasm is obscure; but Bruetsch [22] has found plaques present in the optic chiasm in demyelinating diseases, and it would seem that some cases might be considered under this general heading.

I do not wish to imply that visual loss may not rarely result from a primary arachnoiditis, however. Yuhl and Rand [23] have reported a proved case of tuberculous arachnoiditis involving the chiasmal region in which very dense matted adhesions were present. A complete bitemporal hemianopsia, with visual acuity reduced to 20/200 in each eye, quickly disappeared after surgery, and the vision returned to 20/20 in each eye. This is somewhat the response we should expect if the arachnoiditis is the cause of the loss of vision and the optic discs do not show severe atrophy.

I would suggest that those cases in which surgery uncovers only minimal adhesions and collections of fluid about the chiasm and in which prompt visual improvement does not occur after surgery be referred to as optochiasmic neuritis with or without associated arachnoiditis. With a separation of cases in which the visual loss is the result of a primary process in the optic nerve and/or chiasm from those in which the visual loss is actually due to the adhesions of an arachnoiditis, we may have a better chance to uncover the diverse etiologies which undoubtedly are present in the diseases now grouped under the heading of optochiasmic arachnoiditis.

Fig. 7.—Photograph demonstrating café au lait spots in Case 3.

Case 3.—This patient was a 4-year-old girl who accompanied her older sister to the eye clinic one

§ References 18 and 22.

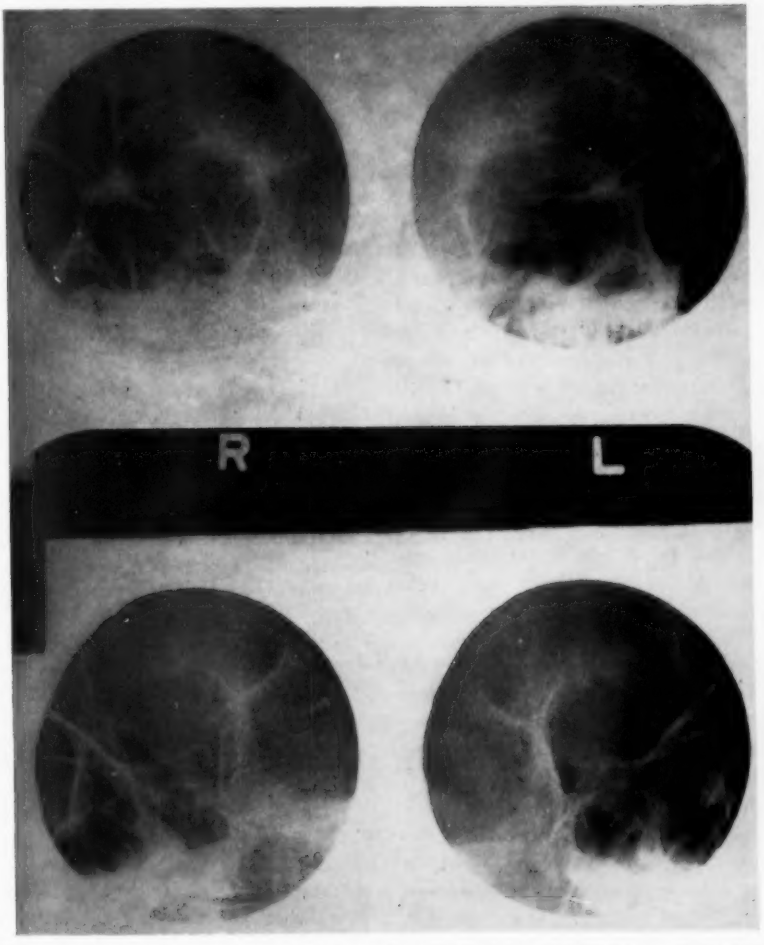

Fig. 8 (Case 3).—X-ray demonstrating bilateral enlargement of optic foramen. For comparison, normal optic foramina are shown above, with those of the patient below.

day. The older sister was the patient, but the mother asked the advice of the resident regarding an exotropia which the younger girl presented. For some reason, instead of simply advising the mother on what course to pursue regarding definitive examination, the resident glanced at the fundi with his ophthalmoscope and noticed the presence of a bilateral primary optic atrophy. This immediately aroused considerable concern, and a complete examination was undertaken. The child was too young for field examination, but visual acuity was found to be 20/175 R. E. and perception of light and motion L. E. Rotations of the eyes were normal, and the exotropia was felt to be secondary to the poor vision.

The child demonstrated several café au lait spots on her body (Fig. 7). The older sister had an extensive hemangioma of the left orbit, cheek, and palate, as well as café au lait spots.

X-rays revealed bilaterally enlarged optic foramina (Fig. 8). A preoperative diagnosis of chiasmal glioma was made. Operation revealed diffuse enlargement of the chiasm and adjacent optic nerves. The left optic nerve was decompressed by unroofing the optic foramen, but the nerve did not appear to be under pressure. A biopsy of the left optic nerve was done. The pathologic report confirmed the diagnosis of glioma, in which the normal optic nerve fibers had been replaced by a diffuse infiltrative syncytium of astrocytic elements. One year after surgery there was no change in vision.

COMMENT

Gliomas of the chiasm represent but about 0.84% of all verified intracranial tumors.[24] In spite of their relative rarity, the diagnosis is not difficult if certain salient features are kept in mind.

Ninety per cent of chiasmal or optic nerve gliomas (they represent essentially the same tumor) occur within the first two decades of life. As Verhoeff [25] has pointed out, they are essentially congenital in origin. Visual acuity is affected early in the course of the disease, owing to replacement of the nerve fibers by a neuroglia syncytium. Since Davis' [26] classic monograph on association of glioma of the optic nerve and chiasm with other evidences of Recklinghausen's disease in the patient or members of his family, the finding of this evidence in a patient suspected of glioma is of great help in the diagnosis. In at least 37% ‖ of patients with optic nerve gliomas, other manifestations of Recklinghausen's disease will be found, and approximately 30% [29] of patients with chiasmal gliomas are said to show other evidence of Recklinghausen's disease. If extremely careful evaluation of the patient for other signs of Recklinghausen's disease were carried out, the percentage would probably be much greater. As Davis ¶ has pointed out in 15 consecutive cases of optic nerve glioma, in every case he has observed other evidence of Recklinghausen's disease. Café au lait spots are the most typical manifestation.

In chiasmal gliomas the x-ray reveals bilateral enlargement of the optic foramina. Proptosis is usually not present in a chiasmal glioma. Gliomas of the optic nerve may or may not present proptosis.

Treatment of chiasmal glioma is unsatisfactory, and our patient was no exception. Fortunately, many gliomas seem self-limiting. Surgery was undertaken in our patient in the hope that some pedunculated extensions could be removed, and also to decompress the optic foramen. This patient is of interest because she presented the classic findings of the relatively rare condition of chiasmal glioma and because of the occurrence of massive hemangioma in her sister.

‖ References 27 and 28.

¶ Davis, F. A., in discussion on Marshall.[27]

SUMMARY

The cases presented illustrate briefly a few of the protean manifestations of chiasmal disease. Emphasis has been placed upon certain features of each case in the diagnosis and classification of chiasmal disease.

203 Doctors' Building.

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Therapeutic Studies in Experimental Chemical Injury of the Cornea

I. Calsulfhydryl (Hydrosulphosol) Studies

GUSTAV C. BAHN, M.D.
and
JAMES H. ALLEN, M.D., New Orleans

This investigation is part of a systematic survey to determine the efficacy of current and newly developed methods of treating chemical corneal injuries. From the gamut of drugs available for use in chemical burns, calsulfhydryl (Hydrosulphosol) was selected for early evaluation because of its relative newness as a therapeutic agent, and because of the wide difference of opinion as to its clinical value. Either chemical or mechanical rupture of cell membranes increases the oxidative activity of the damaged tissue. Supply of a supplementary reducing agent tends to counteract this effect and to restore a more nearly normal oxidation-reduction balance in the aerobic enzyme systems of the corneal epithelium. Free sulfhydryl groups, which contain readily available hydrogen and sulfur, theoretically provide the necessary reductive power. Calsulfhydryl, which contains a fairly high concentration of free sulfhydryl groups, has been recommended in the treatment of almost all types of corneal lesions on the basis of the above concept.

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From the Department of Ophthalmology, Tulane University of Louisiana School of Medicine.

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MATERIALS AND METHODS

A. Animals.—Albino rats of the Wistar strain, weighing approximately 100 gm., were chosen as the experimental animal because their availability in large numbers rendered greater statistical significance to the data which they provided.

B. Solutions.—E. Burn agents

Corneal lesions were produced with representative mineral acids and alkalis, among which were hydrochloric, nitric, sulfuric, and iodoacetic acids and sodium hydroxide, ammonium hydroxide, and calcium oxide. The results of eyes burned with 0.1 N hydrochloric acid and 0.1 N sodium hydroxide and treated with calsulfhydryl are presented at this time.

2. Forms of calsulfhydryl used

(*a*) Concentrate

(*b*) Ophthalmic calsulfhydryl (5% in castor oil)

(*c*) Calsulfhydryl, 5% aqueous solution

3. Pure castor oil U. S. P. (used in one experiment as a control)

C. Method of Study.—1. Standard Lesions: The size of rat cornea narrowed the choice of method whereby a reasonably standard lesion could be produced. After an attempt with the several methods of producing standard burns discussed by Harley,[2] Friedenwald,[1] and others, a modified splash method was adopted. The burn was produced without anesthesia in order to eliminate anesthetic modification of the injury and to duplicate more nearly the conditions under which the clinical injury occurs. The lids of both eyes were retracted, and the burning agent was dropped upon each cornea at the rate of 1 drop every 5 seconds for 30 seconds. The acid or alkali was allowed to remain in contact with the cornea for an additional 30 seconds with the lids closed. Both eyes were then copiously flushed with distilled water for 30 seconds. Distilled water was chosen as the irrigant, since water is most commonly

Fig. 1.—Method of maintaining lid closure. Loop sutures placed in upper and lower lids, with third suture passed through loops.

used clinically for this purpose. Each eye was examined within 10 minutes in order to insure adequate severity of the injury.

2. Preparation of the Treated and Control Eyes: In several of the experiments, maintained lid closure was necessary. This was accomplished by placing a 3-0 blue cotton loop suture just above the lid margin of the upper lid and another loose loop in the region of the orbital rim in the lid inferiorly. Each suture was knotted quadruple. The lids were closed by placing a loop of orange 3-0 thread through the loops in the upper and lower lids, and again knotting four times. Different-colored suture material was used to facilitate removal of the closing suture for instillation of medication and examination (Figs. 1 and 2). The lids were wiped free of dried mucus and residual drug with a moist cotton ball prior to examination and instillation of medicine. In all cases, the right eye of the animal was treated, and the left eye was followed as an untreated control.

D. Grading of Severity of Lesions.—A modification of the Harley adaptation of Friedenwald's scheme of grading lesions was adopted for this series of experiments. Work sheets were prepared for recording by a numerical grading system the results of periodic examinations (Clinical Data Sheet). This plan simplified recording of results and rendered the data amenable to breakdown for further statistical analysis should such treatment subsequently be deemed necessary. Examinations were made at 24-hour intervals.

E. Statistical Treatment.—The average score for the eyes of each group of animals for each examination period was determined. Average differences between the treated and the untreated control eyes were obtained, and the Fisher t test applied. P values were calculated for each group of animals at each examination period. Each experimental group consisted of 15 animals. The results and mathematical treatment of data obtained in nine representative experiments are presented in Tables 1 through 9.

EXPERIMENTAL RESULTS

EXPERIMENT 1.—Both eyes of 15 albino rats were burned with 0.1 N hydrochloric acid. Pure castor oil was instilled into the right eye four times a day. The left eye served as an untreated control. The results are seen in Table 1.

Conclusion.—Pure castor oil had neither a beneficial nor a deleterious effect upon the healing rate of 15 rat corneas burned with hydrochloric acid. Treated and nontreated control eyes healed at approximately the same rate.

EXPERIMENT 2.—Standard burns of both eyes of 15 albino rats were produced with 0.1 N hydrochloric acid. The right eye was flooded with calsulfhydryl-castor oil mixture three times a day during the treatment period. Both eyes remained open during the observation period. Results are tabulated in Table 2.

The control eye of one animal became secondarily infected at the 96-hour examination. The score therefore resulted in an extremely high t value, eliminating the possibility of anything but chance producing the score for that day.

Conclusion.—Instillation of calsulfhydryl-castor oil mixture three times a day without lid closure between treatments did not significantly alter the course of recovery from hydrochloric acid burns of the rat cornea.

Fig. 2.—Method of maintaining lid closure. Third suture tied to maintain snug closure of lids.

CLINICAL DATA SHEET

Animal No.		
Experiment No.		
Animal Weight		
Type of Burn		
Observation Interval		
Date of Experiment		
Opacity:		
Intensity	11	
Area	8	
Less ¼	2	
Less ½	4	
Less ¾	6	
Greater ¾	8	
Density	3	
No opacity	0	
Detect. opacity	1	
Mod. opacity	2	
Opaque	3	
Corneal edema	4	
Corneal slough or ulcer	5	
Stain	1	
Denuded epithel.	2	
Moderate slough	3	
Pronounced slough	4	
Perforation	5	
Vascularization	5	
Superficial	2	
Deep	3	
Conjunctiva	11	
Edema	2	
Congestion	2	
Discharge	3	
Necrosis	4	
Iritis	4	
Miosis and photophobia	1	
Iris congest.; flare	2	
Exudative iritis	3	
Panophthalmitis	4	

TABLE 1.—*Comparison of Castor-Oil-Treated Right Eye with Untreated Left Eye Following Standard Burn of Each Eye with 0.1 N Hydrochloric Acid*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	10.79	9.86	−0.93	2.17	0.05
48	11.71	10.21	−1.50	−2.13	0.05
72	9.29	5.07	−4.22	−2.52	0.05
96	8.00	3.64	−4.36	−2.56	0.05
144	1.21	1.0	−0.21	−1.08	0.1

TABLE 2.—*Comparison of Right Eye Treated with Calsulfhydryl in Castor Oil and Untreated Left Eye Following Standard Burn of Each Eye with 0.1 N Hydrochloric Acid*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	9	9	0	0	
48	0	0	0	0	
	Reburned				
24	9.00	9.13	+0.13	1.00	
48	9.13	9.36	+1.87	1.00	
72	9.13	9.20	+0.07	0.96	
96	0	0.53	+0.53	40.00	

TABLE 3.—*Comparison of Calsulfhydryl-Concentrate-Treated Right Eye with Untreated Left Eye Following Standard Burn of Each Eye with 0.1 N Hydrochloric Acid*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	0.966	0.97	+0.04	+3.94	0.01
48	9.46	7.40	2.06	—2.07	0.05
72	7.53	2.13	—5.40	—4.76	0.01
96	2.60	0.41	—1.50	—0.95	0.1
120	1.40	0.20	—1.20	—0.91	0.1
144	2.73	0	—2.73	—1.98	0.1

TABLE 4.—*Comparison of Right Eye Treated with 5% Aqueous Calsulfhydryl Solution with Untreated Left Eye Following Standard Burn of Each Eye with 0.1 N Hydrochloric Acid*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	9.20	9.13	—0.07	—0.47	0.1
48	11.06	9.53	—1.53	—0.37	0.1
72	8.66	3.26	—5.40	—4.18	0.01
96	6.33	1.66	—4.67	—3.14	0.01
120	5.73	0.73	—5.00	—3.47	0.01
168	5.26	0.73	—5.53	—3.24	0.01
216	4.66	4.00	—0.66	—0.36	0.1

EXPERIMENT 3.—Standard lesions were produced in each eye of 15 albino rats with 0.1 N hydrochloric acid. The right eye was treated with calsulfhydryl concentrate three times daily. The left eye received no treatment. Both eyes remained open during the course of healing. Results are tabulated in Table 3.

Conclusion.—Corneal lesions produced by 0.1 N hydrochloric acid healed more slowly when treated with calsulfhydryl concentrate three times daily than did similarly burned untreated eyes. Under the conditions of the experiment, greater residual corneal opacification due to scarring occurred in the eyes treated with calsulfhydryl.

EXPERIMENT 4.—A standard lesion was produced in each eye of 15 albino rats with 0.1 N hydrochloric acid. The right eye was treated with 1:20 aqueous calsulfhydryl solution three times daily. The left eye received no treatment, and both eyes remained open throughout the observation period. Results are shown in Table 4.

Conclusion.—During the first 24 hours following corneal injury by 0.1 N hydrochloric acid, no significant difference was observed between eyes treated with 5% aqueous calfsulfhydryl solution and untreated, control eyes. At the 72-hour examination the treated eyes were more severely damaged than those which had not received treatment. Residual scarring and opacification were significantly greater in the treated eyes.

EXPERIMENT 5.—Bilateral standard corneal lesions were produced in 15 albino rats with 0.1 N hydrochloric acid. Prior to injury, both eyes were prepared for lid closure, as previously described. The right eye was flooded with calsulfhydryl concentrate immediately after injury, and subsequently three times daily. The left eye received no treatment other than lid closure. The eyes were examined at 24-hour intervals. Results are shown in Table 5.

TABLE 5.—*Comparison of Calsulfhydryl-Concentrate- and Lid-Closure Treatment of Right Eye with Lid-Closure Treatment of Left Eye Following Standard Burn of Each Eye with 0.1 N Hydrochloric Acid*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	10.13	10.13	0	0	1.0
48	10.27	9.46	—0.81	—1.85	0.1
72	11.33	9.2	—2.13	—3.39	0.01
96	10.93	6.53	—4.40	—3.98	0.01
120	10.66	4.13	—6.53	—5.08	0.001
144	11.86	2.53	—9.33	—5.46	0.001
360	12.13	0.46	—11.67	—15.08	0.001
404	10.33	0	—10.33	—4.51	0.001

TABLE 6.—*Comparison of Right Eye Treated with Calsulfhydryl-Castor Oil Mixture and Untreated Left Eye Following Standard Burn of Each Eye with 0.1 N Sodium Hydroxide*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	16.40	9.27	−7.13	−5.60	0.001
48	7.53	3.13	−4.40	−2.30	0.05
72	6.00	1.80	−4.20	−4.23	0.001
96	1.92	2.11	+0.29	+0.50	1.0
120	2.00	0.64	−1.36	−2.55	0.05

Conclusion.—During the first 48 hours following corneal injury due to 0.1 N hydrochloric acid, no significant difference was observed between the treated and the untreated eyes. Subsequently, the treated eyes healed more slowly and exhibited greater residual opacification and scarring.

EXPERIMENT 6.—Corneal lesions were produced in 15 albino rats with 0.1 N sodium hydroxide. The right eye was flooded with calsulfhydryl-castor oil mixture three times a day during the observation period. The left eye received no treatment, and both eyes were allowed to remain open. The results are shown in Table 6.

Conclusion.—Rat corneas injured with 0.1 N sodium hydroxide healed more slowly and with greater residual scarring and opacification when treated with calsulfhydryl and castor oil than did similarly damaged untreated eyes. At the 96-hour examination, the score for the treated eyes was slightly lower than that for the untreated eyes. No statistical significance was attached to this difference, however.

EXPERIMENT 7.—Standard corneal lesions were produced by 0.1 N sodium hydroxide in 15 albino rats. The right eyes were treated by instillation of calsulfhydryl concentrate three times daily. No treatment was provided the left eye. The eyes were allowed to remain open during the observation period. The results are presented in Table 7.

Conclusion.—During the first 96-hour period following injury, no significant difference was observed between eyes treated with calsulfhydryl concentrate and the untreated, control eyes. Subsequently, the treated eyes healed with significantly greater opacification.

EXPERIMENT 8.—Both eyes of 15 albino rats were burned with 0.1 N sodium hydroxide. The right eye was treated with calsulfhydryl concentrate, the left eye serving as an untreated control. The eyes were not closed during the observation period. The results are shown in Table 8.

TABLE 7.—*Comparison of Calsulfhydryl-Concentrate-Treated Right Eye with Untreated Left Eye Following Standard Burn of Each Eye with 0.1 N Sodium Hydroxide*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	24.80	23.13	−1.67	−3.35	−0.01
48	26.53	23.80	−2.73	−1.18	0.1
72	30.53	25.33	−5.20	−1.90	0.1
96	30.40	26.73	−3.67	−1.15	0.1
144	34.40	27.67	−6.86	−2.37	0.05
168	37.33	28.48	−8.86	−3.57	0.01
192	38.60	31.20	−7.40	−2.94	0.01
216	38.60	33.20	−5.40	−2.52	0.01
264	38.07	33.90	−4.17	−2.04	0.05

TABLE 8.—*Comparison of Calsulfhydryl-Concentrate-Treated Right Eye with Untreated Left Eye Following Standard Burn of Each Eye with 0.1 N Sodium Hydroxide*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	12.60	1.40	−11.20	−5.67	0.001
48	15.53	1.00	−14.53	−6.06	0.001
72	14.60	1.80	−12.80	−5.34	0.001
96	14.27	0.53	−13.74	−6.06	0.001
120	12.87	1.00	−11.87	−4.94	0.001
240	13.46	3.00	−10.46	−3.71	0.01
360	13.20	2.06	−11.14	−3.83	0.01

TABLE 9.—*Comparison of Calsulfhydryl and Lid-Closure Treatment of Right Eye with Lid-Closure Treatment of Left Eye Following Standard Burn of Each Eye with 0.1 N Sodium Hydroxide*

Hours Following Injury	Average Score Treated Eye	Average Score Control Eye	Average Difference	t	P
24	17.40	4.05	−13.33	−7.44	0.001
48	19.77	6.53	−12.74	−6.80	0.001
72	20.20	7.20	−13.00	−3.73	0.01
96	20.40	5.87	−14.53	−6.08	0.001
120	17.93	7.26	−10.17	−2.65	0.02
240	15.73	7.33	− 8.40	−1.80	0.1
504	13.67	5.73	− 7.94	−1.85	0.1

Conclusion.—Rat corneas injured with 0.1 N sodium hydroxide and treated with calsulfhydryl concentrate remained inflamed longer and healed with more residual opacification than did similar injured untreated eyes. Calsulfhydryl was found to have a deleterious effect under the conditions of the experiment.

EXPERIMENT 9.—Both eyes of 15 albino rats were burned with 0.1 N sodium hydroxide solution. Prior to injury, lid sutures were placed in order that closure could be maintained during the observation period. Immediately following the injury, the right eye was flooded with calsulfhydryl concentrate. The lids were closed and reopened only for examination or treatment. Examinations were made at 24-hour intervals. The results are shown in Table 9.

Conclusion.—Rat corneas burned with 0.1 N sodium hydroxide and treated with calsulfhydryl concentrate three times daily and lid closure failed to heal as rapidly and completely as did similarly injured corneas which received only lid closure as treatment. Calsulfhydryl had a significantly deleterious effect upon rat corneas under the conditions of this experiment.

SUMMARY

In a series of four experiments in which rat eyes were burned with 0.1 N hydrochloric acid and treated with several forms of calsulfhydryl (Hydrosulphosol), the drug proved to have no beneficial effect. In another series of four experiments in which rat eyes were burned with 0.1 N sodium hydroxide and treated with several forms of calsulfhydryl, the drug proved to have either no beneficial effect or a definitely deleterious effect. Subsequent reports will include data obtained in experiments employing other burn agents.

Dr. George W. Gibbins assisted in Experiments 1 through 4, on acid burns of the cornea.

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The Etiology of Retinochoroiditis and Uveitis

Importance of the Dye (Methylene Blue) Cytoplasm-Modifying Antibody Test for Toxoplasmosis

J. V. CASSADY, M.D.
CARL S. CULBERTSON, M.D.
and
JAMES W. BAHLER, M.S., South Bend, Ind.

Since Wilder [1] found Toxoplasma-like organisms in many chorioretinal eye lesions at the Armed Forces Institute of Pathology, the significance of toxoplasmosis as a cause of chorioretinitis has assumed major importance. The causative agent of toxoplasmosis (Toxoplasma gondii) is a protozoan which was discovered by Nicolle and Manceaux in 1908.[2] The lunate shape of the organism accounts for its name, which is derived from the Greek τόξον, meaning a bow, or arc. It was first found in the gondi, a small North African rodent used in the laboratory of the Pasteur Institute in Tunis. That this parasite would produce human infections was discovered by Wolf, Cowen, and Paige,* and also by Sabin,[5] in 1939.

Cross,[6] in a cytologic study of Toxoplasma, showed that in various stages of growth the spherical forms elongate and undergo binary fission and that this always occurs intracellularly. When the host cells are no longer able to supply nourishment, growth stops. During this resting stage, the nucleus of the host cell may be lost, and the wall of the terminal colony may resist rupture and produce a resting or resistant state, in which the parasites remain inactive for an indefinite length of time.

During this stage, there is no inward penetration of host antibodies and no outward escape of the metabolic wastes of the parasites to induce local inflammatory reaction. The wall of the terminal colony, however, may rupture and free the enclosed parasites.

Wolf and Cowen,[7] in 1937, pointed out that Janku [8] had probably reported the first instance of human toxoplasmic infection of the choroid and retina. He had found parasites (probably Toxoplasma) in the retina but was unable to identify or classify them. The recognition of chorioretinitis caused by Toxoplasma in 1943 by Koch, Wolf, Cowen, and Paige [9] in six cases of infantile toxoplasmic encephalomyelitis started a rather premature enthusiasm about toxoplasmosis among ophthalmologists. Vail [10] and Vail, Strong, and Stephenson [11] reported six cases of chorioretinitis as probably due to Toxoplasma because they had neutralizing antibodies against this organism. Heath and Zuelzer, [12] in 1944, found chorioretinitis in both eyes of twins, in one of which autopsy showed lesions in its brain, liver, and spleen, as well as chorioretinitis caused by Toxoplasma. Wagener,[13] in 1944, in reviewing the literature of toxoplasmic chorioretinitis, was of the opinion that the disease was relatively common and that its diagnosis was not too difficult. The diagnosis of toxoplasmic chorioretinitis is, however, quite Herculean. For several years it has been recognized that infantile chorioretinitis might be caused by prenatal intrauterine infection. The affected infants frequently have mental retardation, cerebral calcification, or other neurologic signs to corroborate the etiologic diagnosis. Frenkel [14] assumed that an encysted, quiescent, resting terminal colony of Toxoplasma starting in utero or as an infantile toxoplasmosis could persist in the retina, choroid, or ciliary body and later rupture to incite a childhood or an adult flare-up of toxoplasmic chorioretinitis.

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* References 3 and 4.

Pinkerton and Henderson,[15] in 1941, recognized one of the first fatal cases of adult toxoplasmosis. Since then, quite a few reports of unquestioned acquired infections of adults by Toxoplasma have been reported, some in laboratory workers. They showed a clinical picture of fever, pneumonitis, hepatitis, and lymphadenopathy, and some exanthematous rashes. In most of these adult infections no ocular involvement was observed. Hogan,[16] in 1947, was inclined to accept the clinical picture of toxoplasmic chorioretinitis, but he believed then that congenital toxoplasmosis was the only form that exhibited ocular lesions.

In 1948, Sabin and Feldman [17a] described the cytoplasm-modifying antibody test, using methylene blue dye. Sera containing antibodies against Toxoplasma prevented the staining by alkaline methylene blue dye of the cytoplasm of Toxoplasma organisms freshly harvested from mouse peritoneal exudate. This test, hereinafter referred to as the dye test, constitutes a milestone in progress against this infection.

Sabin and Feldman [17b] warned against the clinical diagnosis of toxoplasmosis because a large number of children who clinically seemed to have toxoplasmosis (with chorioretinopathy and cerebral damage) had negative serologic tests for this disease. If there was an associated cerebral calcification, the chances were good of its being associated with congenital toxoplasmosis. Sabin,[18] in 1950, believed that chorioretinitis could be due to toxoplasmosis only if the disease was of congenital origin, but never if it was acquired, especially by adults. Walsh,[19] Hogan,[20] and Sabin,[21] in a symposium on toxoplasmosis, described chorioretinitis of toxoplasmosis as being in the posterior pole of the eye, usually bilateral, and associated with nystagmus, optic atrophy, convulsions, cerebral calcification, hydrocephalus, or mental deficiency. The child's infection is transmitted from the mother, but how she acquires it is not clear. Sabin, however, was very reluctant to accept the possibility of acquired toxoplasmosis at any age and considered even congenital toxoplasmosis as being not nearly so often the cause of chorioretinitis as ophthalmologists were prone to believe.

From the time that toxoplasmosis was first found to be an etiologic factor in chorioretinitis, ophthalmologists very enthusiastically welcomed it as a new causative agent for many hitherto unexplained cases of posterior uveitis. The presumptive diagnosis of tuberculous chorioretinal pathology was often difficult to accept. At the 1950 Academy meeting, however, Sabin cooled the enthusiasm for Toxoplasma until most ophthalmologists considered it an unlikely factor in the etiology of retinochoroiditis. Then, in April, 1952, Wilder [22] reported 41 cases of chorioretinitis in which Toxoplasma-like organisms were found microscopically at the Registry of Ophthalmic Pathology in Washington; and in August, 1952, she reported finding additional cases of Toxoplasma-like organisms in lesions of retinochoroiditis in patients ranging from 14 to 83 years of age at the time of enucleation of the eye. Serologic tests for toxoplasmosis on many of these patients were done by Woods, Jacobs, Wood, and Cook [37] and confirmed the histologic diagnosis of toxoplasmic retinochoroiditis. This discovery again stimulated interest in toxoplasmosis as a cause of chorioretinitis. Wilder reported that among 131 eyes with the pathologic diagnosis of tuberculosis, Toxoplasma-like protozoa were demonstrated in 30, whereas in only 3 were tubercle bacilli found, although all of them had been subjected to a careful search after acid-fast staining.

Hogan, Thygeson, and Kimura,[23] in 1952, reviewed 338 cases of uveitis which they had studied for a possible toxoplasmosis etiology. They found that the diagnosis and proof of a Toxoplasma etiology for uveitis are very difficult because the normal population has serum antibodies against Toxoplasma. They urged that tests of serum for changing titer should be made during different phases of uveal tract disease. Duke-Elder,[24] in 1953, reported finding Toxoplasma in an enucle-

ated eye of a man who had been treated for years with recurring attacks of chorioretinitis believed to have been tuberculosis, and whose eye was finally removed because it was blind and painful. In this eye, Toxoplasma-like protozoa were found in necrotic areas of the retina and in nodular lesions of the iris. A search of 32 eyes in which the diagnosis of granulomatous uveitis had been made in the Duke-Elder Clinic revealed that about half of them showed structures clinically resembling Toxoplasma protozoa. In a symposium on toxoplasmosis in 1953 by Sabin,[25] Jacobs,[26] Frenkel,[27] Wilder,[28] Eyles,[30] and Feldman,[29] the subject was reviewed again. The explanation offered for congenital toxoplasmosis was that the fetus was infected only if the mother had acquired her infection during the gestation period. Depending upon the particular trimester during which the pregnant mother acquired toxoplasmosis, the fetus may show an active encephalomyelitis, and/or retinochoroiditis, either of which may be active or healed. Those who give birth to infected children invariably exhibit the high antibody titers expected of recent infection. A possibility of the transfer of the Toxoplasma to the infant by infected mother's milk was also suggested.

Cole, Prior, Docton, Chamberlain, and Saslaw [31] reviewed the relationship of toxoplasmosis to families that had had contact with dogs proved to have been infected with Toxoplasma. Toxoplasma parasitemia was found in one woman who had cared for a dog dying of toxoplasmosis. Two dogs contracted the disease from a 3-year-old boy with toxoplasmic chorioretinitis and encephalitis. Koke [32] reported an instance of a mother that had toxoplasmic chorioretinitis two weeks before the birth of a child. Both she and the child had retinochoroiditis and a positive dye test for toxoplasmosis. Woods, Jacobs, Wood, and Cook [33] reported on some 400 patients upon whose sera the dye test for evidence of toxoplasmosis had been performed. These included 201 patients with granulomatous uveitis, 110 with nongranulomatous uveitis, and 98 nonuveitis controls. They adopted the following criteria used by Beverley and Beattie [34] for positive dye tests in the various age groups:

Age, Yr.	Positive Titer
Below 10	1:8 or over
10 to 19	1:32 or over
Over 20	1:64 or over

In old quiescent cases of probable ocular toxoplasmosis, the dye test was usually of moderate titer (1:64 to 1:256). Ocular findings in the cases studied by Woods were retinochoroiditis, granulomatous uveitis, retinal periphlebitis, and optic neuritis. In this series, 58 of 201 cases of granulomatous uveitis had significantly positive methylene blue dye tests, indicating a toxoplasmic etiology. In a group where the previous medical survey had failed to reveal any etiology to explain the ocular disease, 56% had positive dye tests. Woods concluded that a Toxoplasma infection is the probable etiologic factor in 25% of cases of adult granulomatous uveal disease. On the basis of these findings, it would seem that toxoplasmosis in the adult is a widespread and extremely common infection, although only rarely showing any systemic evidence of infection. The parasite apparently has an affinity for ocular tissue, the eyes appearing more susceptible to toxoplasmosis.

Jacobs, Fair, and Bickerton [35] isolated live Toxoplasma parasites from an enucleated human eye of an adult with chorioretinitis and were able to infect mice and tissue cultures with the organism. The dye test titer of the patient was only 1:64 before enucleation, but rose to 1:4096 shortly after. This case definitely establishes adult ocular toxoplasmosis as a disease entity. Ryan, Hart, and associates [36] reported a large number of patients who had had negative clinical surveys for other possible causes of their eye disease. They used pyrimethamine and sulfadiazine to treat their patients and found that this therapeutic regimen seemed to be helpful. Cortisone given systemically aggravated the ocular disease in some cases, unless it was given with anti-Toxoplasma therapy. Passmore,[39] early this year, reviewed the

case histories of 13 adult patients with toxoplasmic granulomatous uveitis, some of whom were benefited by pyrimethamine therapy.

THE DYE TEST FOR TOXOPLASMOSIS

The superiority of the dye (methylene blue) cytoplasm-modifying antibody test of Sabin and Feldman over other diagnostic procedures in Toxoplasma infection has been repeatedly emphasized. The facilities for the performance of this test, however, are not easily available. During the early part of this study, dye tests were performed by the Communicable Disease Center of the United States Public Health Service at Chamblee, Ga.

Because of difficulties having to do with preservation of the specimens, coordination of clinical information, and shipping problems, and because of the constantly increasing number of tests which were considered necessary in the study, facilities for performance of the test were made available locally by the South Bend Medical Foundation. The tests are time-consuming, and they require a continuous passage of the parasite in the peritoneal cavity in mice in order to keep a necessary supply of living Toxoplasma organisms for the test. Since the cost for each test would be prohibitive for many private patients, the tests were performed as a research project. In those tests performed in South Bend, the modifications of Beverley and Beattie were employed. The RH strain of Toxoplasma was obtained through Dr. William Summers, of Indiana University Medical Center, Indianapolis, and Dr. Max Cowan, of the Eli Lilly Research Laboratories, Indianapolis. Peritoneal exudate from 18- to 20-gm. mice inoculated three days previously provided Toxoplasma organisms in sufficient numbers for the tests. Preliminary studies on sera of known antibody levels served as standard controls for the method. Search for sera of negative antibody content for use as "activator" provided experience in studying the "end-point" of the test. The results of the test in each case are reported in the individual case descriptions.

Until April, 1955, a third of the dye tests for toxoplasmosis done locally, or 44 tests, were done on 36 of our own patients who had either inflammatory or circulatory ocular disease. The sera of one-third of the 36 were negative or were positive only in a dilution of less than 1:8. Two-thirds had positive dye tests of a titer of 1:32 or higher. Corroborative evidence makes it seem likely that 23 of these 36 patients had Toxoplasma infection. The ocular disease in 16 of these patients was active and in 7 was healed, or at least quiescent.

CLINICAL EXPERIENCE IN STUDYING ETIOLOGY OF GRANULOMATOUS UVEITIS

The present study is based upon clinical experience with 23 adult patients who have had active granulomatous retinochoroiditis or comparable ocular disease. This group of patients, in practically every instance, were hospitalized for clinical and serologic study to determine the cause of their ocular disease. The clinical survey, done by internists, was to rule out systemic disease, such as diabetes, sarcoidosis, chronic nephritis, leukemia, or tuberculosis. Foci of infection, such as sinuses, teeth, and tonsils, were searched for by otolaryngologists and dentists. The serologic tests performed were those for Brucella abortus and Pasteurella tularensis, V. D. R. L. tests, etc. Roentgenograms of the chest, sinuses, and skull, and, where indicated, the hands and feet, were routinely obtained to rule out active or healed pulmonary pathology, first-infection tuberculosis, cerebral calcification, sinus disease, or any changes associated with sarcoidosis. PPD intradermal tests were done, in dilutions of 1:10,000 or 1:1000 or both, and in some instances 1:100, on all these patients. Toxoplasmin skin tests were done in about half these cases, but usually not until after the blood had been drawn for the cytoplasm-modifying antibody test.

Since the dye test has been available, 16 patients have been encountered (Table 1) on whom negative clinical and serologic studies (as described above) have left only a toxoplasmic etiology to account for their ocular

TABLE 1.—*Data on Sixteen Patients with Active Ocular Disease, Whose Dye Test for Toxoplasmosis Was Positive and Whose Clinical, Serologic, and Roentgenologic Surveys Were Negative for Any Other Etiology* *

Initial	Age	Sex	Eye Lesion *	Previous Attacks	Positive Dye Test Titer	Treatment	Improvement
R. A.	34	M	R. C.; Ac. ex.	11 yr.	1:512	Yes	Moderate
D. A.	34	F	Gran. U. 5 mo.	0	1:64 1:32 1:32	Yes	Marked in 1 wk.
R. B.	32	F	Bl. I.; Gran. U.; Ac. ex. 2 mo.	Every year for 13 yr.	1:512	Yes	Marked in 1 wk.
H. C.	43	M	Bl. I.; R. C.; Ac. ex. 2 mo.	Every year or 2 since 24 yr. of age	1:64 1:128	Yes	Plasmocytopenia and leucopenia
D. D.	26	F	R. C.; Ac. ex. 4 mo.	None	1:16 1:128	Yes	Marked in 1 wk.
J. M.	22	F	R. C.; Ac. ex. 18 mo. duration	None	1:64	None	Still slight activity
R. M.	16	M	R. C.; central Ac. ex.	None	1:256	None	V-20/200 None
H. H.	25	F	R. C.; Ac. ex. (pregnancy)	5 yr. before (pregnancy)	1:64 1:32	None	Persisted 5 mo.
R. H.	26	M	Papillitis; optic neuritis	None	1:64	None	Subsided
C. P.	30	M	R. C.; Ac. ex.	2 yr. previously	1:256	Yes	Marked, but detachment was found
H. P.	32	M	Gran. U. 1 mo.	Every few mo. —6 yr.	1:128 1:256	Yes	Marked, 1 wk.
W. R.	26	M	R. C.; Ac. ex. 5 mo.	10 yr. previously	1:256 1:128 1:512	Yes	Marked, 1 wk.
J. S.	50	F	R. C.; Ac. ex.; Gran. U.	None	1:256 1:512	Yes	Marked
A. S.	54	M	R. C.; Ac. ex.; Gran. U.	None	1:128	Yes	Moderate
S. Z.	30	F	R. C. + Gran. U., severe	None	1:128	Yes	Moderate

* R. C. indicates retinochoroiditis; Gran. U., granulomatous uveitis; Ac. ex., acute exacerbation, and Bl. I., blind eye from previous choroiditis.

lesions. In this group of patients, the dye test titers varied from 1:64 to 1:512 and their toxoplasmin skin tests were positive. Their ages varied from 16 to 50 years. When the fundus was visible, the usual ocular disease found was retinochoroiditis, but in some cases the vitreous was so cloudy that the diagnosis could not be more specific than granulomatous uveitis.

The ocular disease in these patients does not represent a recent acute infection with toxoplasmosis, but is a chronic disease with reactivation of quiescent granulomatous uveitis. In some the retinal lesions may be new, resulting from the release of encysted organisms, which incite exudative necrosis of the retina. The accompanying granulomatous choroiditis is a secondary reaction, which extends as an endophthalmitis to the adjacent sclera. Frenkel's description of encysted toxoplasmas in a dormant state explains long periods of inactive retinochoroiditis, which flares up sometimes years after the primary infection.

Tables 1 and 2 list the sex, age, ocular disease, history, dye test titer of the patient's sera, their treatment, and the end-result in these 23 patients. Although the treatment was individualized, it consisted of pyrimethamine (2,4-diamino-5-[*p*-chlorophenyl]-6-ethyl pyrimidine, manufactured under the trade name of Daraprim), sulfadiazine, and hydrocortisone. The dosage of the pyrimethamine was 25 mg. per 60 lb., and that of the sulfadiazine 1 gm. per 60 lb., in 24 hours. The hydrocortisone was given in 20 mg. tablets four times daily. Platelet and white blood cell counts and hemoglobin determinations were done at first at three-day intervals, and later weekly if the medication was tolerated. Intensive treatment was used for a week or 10 days; then the dosage was reduced about half. If possible, the pyrimethamine was given daily for at least a month.

This treatment was given to 10 patients, in 1 of them with nearly disastrous results. The white blood cell and platelet counts were greatly depressed in this patient, and the bone marrow studies showed marked maturation arrest. This patient recovered, however, after administration of corticotropin by slow intravenous drip. It is extremely dangerous to use pyrimethamine without adequate blood studies; however, when the drug is properly used, the effect upon toxoplasmic ocular disease is remarkable. The vitreous clears; the activity of the uveitis subsides; the retinal lesion becomes pigmented, and in most instances within a week after the pyrimethamine and sulfadiazine is started, improvement is apparent.

In Table 2, seven case histories are summarized in which the retinochoroiditis was quiescent, but when it had been active, the same exhaustive search for its etiology had been done, with entirely negative results. When the dye test for toxoplasmosis became available locally, these patients' histories were again reviewed and their sera were tested by the cytoplasm-modifying antibody test for toxoplasmosis. Their dye tests were positive in a titer of 1:32 except in one instance, in which the titer was 1:64. Since investigation for any other etiologic factor was entirely negative, the positive dye test even in this titer (1:32) probably is significant.

The detailed case histories of three of these patients that received antitoxoplasmosis therapy are described.

REPORT OF CASES

Case 1.—J. S., a woman aged 50, April, 1954.

Complaint: Blurred vision, left eye, six weeks.

Examination: Retinochoroiditis, left eye; cloudy vitreous; 20/200 vision.

After systemic cortisone treatment, severe iridocyclitis developed with such pain that hospitalization was required. A clinical and dental survey for an etiologic factor was unrevealing. A 1:1000 Mantoux test was negative. Roentgenograms of the chest were negative. The patient was treated systemically with streptomycin, isoniazid, and PAS for a month, without improvement of the uveitis. The dye test for toxoplasmosis was positive in a dilution of 1:512. One week after start of pyrimethamine, sulfadiazine, and hydrocortisone systemically, the vision improved to 20/40 in the affected eye, with such marked clearing of the vitreous that the fundus became visible. An area of active retinochoroiditis and one area surrounded by pigment could then be seen in the extreme periphery of the fundus. The antitoxoplasmosis therapy was continued for a month, with complete subsidence of the uveitis. There has been no recurrence.

Case 2.—R. B., a woman aged 32; October, 1942.

Complaint: Bilateral uveitis since May, 1941.

Examination: Vision 20/100 and 20/200. Bilateral iridocyclitis and posterior synechiae.

In the left eye complete posterior synechiae could not be broken by mydriatics. Clinical, dental, roentgenographic, and serologic surveys revealed the only significant finding, a mildly positive Mantoux test in 1:100 dilution. Roentgenograms of her lungs showed a Ghon tubercle, but nothing else. The iridocyclitis persisted until complete posterior synechiae, pupillary membrane, and a band-shaped area of corneal degeneration remained. In August, 1954, she was seen again because of acute uveitis and iritis in her right eye. Posterior synechiae in this eye could not be broken, and the fundus could not be seen. After a month's treat-

Table 2.—*Data on Seven Patients with Quiescent Ocular Disease, Whose Dye Tests Were Significantly Positive, and Whose Clinical, Serologic, and Roentgenologic Surveys Were Negative for Any Other Etiology* *

Initial	Age	Sex	Eye Lesion	Age at Previous Attacks, Yr.	Positive Dye Test Titer
J. T.	39	M	Cen.; R. C.; H.	27 14	1:32
E. D.	27	F	Cen.; R. C.; H.	17 20	1:32
V. A.	44	F	Cen.; R. C.; H.	14	1:32
C. R.	21	M	R. C.; H.; M.	22	1:32
A. M.	39	F	R. C.; M.	None	1:32
J. M.	22	F	R. C.; H. Recently active	19	1:32
F. V.	33	M	R. C.; H.; M.	30 28	1:64

* R. C. indicates retinochoroiditis; Gran. U., granulomatous uveitis; Cen., central; H., healed, and M., multiple.

ment with streptomycin, PAS, and isoniazid, the uveitis was worse. In September, the methylene blue dye test for toxoplasmosis was positive in 1:512 titer. Within a week after pyrimethamine, sulfadiazine, and hydrocortisone therapy was started, the uveitis subsided, the pupil could be dilated, and several areas of healed and one area of unhealed retinochoroiditis were visible. Treatment was continued for a month, and since then there has been no recurrence.

CASE 3.—D. D., a girl aged 16; March, 1954.

Complaint: Blurred vision, right eye, one week.

Influenza had preceded the blurred vision. There was an area of acute retinochoroiditis above and temporal to the disc, consisting of a white, oval-shaped patch of edema of the retina with vitreous cloudiness. Her clinical, dental, serologic, and roentgenographic survey was negative except for a 2500 leucocyte count with a normal differential count. Bone marrow, as well as peripheral blood, studies were negative for L. E. cells. The Mantoux test was negative. A cytoplasm-modifying antibody test for toxoplasmosis was positive in 1:16 dilution on April 10, 1954, but was positive in 1:128 titer Sept. 14, 1954. By June her WBC count was 5000, with a normal differential count. The uveitis remained active until September, and the cloudy vitreous reduced her vision to 20/80. After report of the positive dye test in 1:128 titer, she was treated with pyrimethamine, sulfadiazine, and hydrocortisone. At the end of one week, the vitreous cleared, until the vision was 20/30. The treatment was continued for a month, by which time the lesion was pigmented and healed. There has been no recurrence, and the blood cell count and platelet counts were not affected.

COMMENT

A review of some of the literature on toxoplasmosis affirms the almost universal prevalence of the disease in man and animals, its affinity for nerve tissue, its resistance during the encysted stage, its responsibility for encephalitis and retinitis in some infants, the difficulty of being sure it is the cause of ocular disease in adults, and our failure to understand its transmission. Because of its widespread presence in almost all the adult population, Sabin, Feldman, and others have been reluctant to accept it as a cause of ocular disease. Sabin and Feldman made the most important contribution to our recognition of this disease, the cytoplasm-modifying (dye) antibody test. Wilder, Duke-Elder, Woods, Jacobs, Frenkel, their associates, and others have presented strong evidence, in spite of earlier teaching to the contrary, that adult ocular disease is often due to toxoplasmosis. Its frequency as an etiologic factor is now being realized.

Because the cytoplasm-modifying antibody test for toxoplasmosis was available locally, it was possible to study cases of retinochoroiditis and uveitis to determine if toxoplasmosis was a possible etiologic factor. The ocular lesions presumed to be caused by Toxoplasma were uveitis, iridocyclitis, and retinochoroiditis, either fresh local lesions or an activation adjacent to an old healed retinochoroiditis. Clinical and serologic studies of these patients to determine the cause of their ocular disease were almost entirely negative for other causes of their uveitis, but a number of patients were found to have significantly positive cytoplasm-modifying antibody tests for toxoplasmosis.

The toxoplasmic origin of retinochoroiditis or uveitis in these cases is presumptive. But in the past, when there was a positive Mantoux test, we have deduced that the retinochoroiditis was tuberculous, although we did not have positive proof of such an etiology. The methylene blue dye test titer in all of these 23 cases was sufficiently high for a diagnosis of toxoplasmic disease. When this diagnostic test (the dye test) is not readily available, ophthalmologists often look askance at such a high incidence of toxoplasmic etiology. But where these tests are easily obtained, as in this series, toxoplasmosis seems to be a relatively frequent cause of retinochoroiditis. Woods and Jacobs, Ryan and Hart, and associates, and Passmore recently emphasized this fact. It would seem that we should revise our attitude that acquired toxoplasmosis in the adult is symptomless and does not cause eye disease.

In two of these patients (J. S. and W. R.) systemic administration of cortisone aggravated the inflammation of the eye. Ryan and Passmore also noted this peculiarity of toxoplasmic uveitis treated with cortisone, i. e., that whereas most eye inflammations were

allayed by cortisone, the toxoplasmic form on occasion was intensified. A chronic, prolonged, recurring inflammation was characteristic of almost every one of these cases of toxoplasmic uveitis. Tuberculous uveitis also is protracted, but not to such an extent as is this ocular disease. We are hopeful that the severity and duration of the uveitis may now be influenced favorably by pyrimethamine and sulfadiazine, which was so effectively used in some of the present cases. In differentiating whether the etiologic agent is tuberculosis or toxoplasmosis, the therapeutic response may be helpful.

SUMMARY

The sera of 36 patients with vascular or inflammatory disease of the posterior uvea were examined with the serum-modifying antibody dye test of Sabin and Feldman. The sera of 23 of these patients were positive for toxoplasmosis, while a survey for any other etiologic cause for their ocular inflammation was negative. In 7 patients the disease was inactive or quiescent, while in 16 it was active. Eleven patients with active granulomatous uveitis were treated with pyrimethamine and sulfadiazine systemically, producing a marked and dramatic improvement in the uveitis. The treatment may be dangerous and should be used only when blood studies are carefully followed.

Retinochoroiditis or granulomatous uveitis may be caused by tuberculosis or toxoplasmosis, but accusation of either must be presumptive. The appearance of the lesion; the clinical, serologic, and diagnostic tests, and the duration and severity of the lesion must be considered. If the serum-modifying antibody test is readily available, toxoplasmosis as an etiologic cause of eye disease in adults, as well as in infants, will be found more frequently that we formerly believed.

In order that uveitis may be managed intelligently, the treatment must be directed to its cause. If such a high percentage of retinochoroiditis and granulomatous uveitis is due to toxoplasmosis, a complete etiologic survey is not possible without the cytoplasm-modifying antibody dye test of Sabin and Feldman. This test should be made more readily available.

921 Lincoln Way East.

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Phacolytic Glaucoma

A Clinicopathologic Study of One Hundred Thirty-Eight Cases of Glaucoma Associated with Hypermature Cataract

MILTON FLOCKS, M.D., San Francisco
C. STUART LITTWIN, Teaneck, N. J.
and
LORENZ E. ZIMMERMAN, M.D., Washington, D. C.

More than half a century has elapsed since Gifford[1] first described glaucoma due to hypermature cataract and urged its prevention by cataract extraction. Verhoeff and LeMoine,[2] H. Knapp,[3] Kaufman,[4] Courtney,[5] Heath,[6] the Irvines,* Sugar,[10] Hubbersty and Gourlay,[11] and, recently, Ballen and Hughes[12] have reported on their experiences with this entity and have discussed its prevention and appropriate therapy. Theirs have all been reports based on small numbers of cases, and the condition is generally considered to be "unusual."

Glaucoma of this type, which usually has a violent onset, presents a remarkably bland, but strikingly characteristic, pathologic picture: liquefaction of the lens cortex, open iridocorneal angles, and the presence of large histiocytes which have engulfed liquefied lens material and are obstructing the trabecular apparatus. These features were first described by Zeeman,[13] who named the condition "phacogenetic glaucoma." Irvine and Irvine * amplified Zeeman's description, and they and others have emphasized the curability of this glaucoma by cataract extraction.

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Formerly Heed Fellow in Ophthalmic Pathology, Armed Forces Institute of Pathology (Dr. Flocks); present address: Division of Ophthalmology, Stanford University School of Medicine. Formerly Fellow in Ophthalmic Pathology, Armed Forces Institute of Pathology (Dr. Littwin); present address: 950 Queen Anne Rd., Teaneck, N. J. Chief, Section on Ophthalmic Pathology, Armed Forces Institute of Pathology (Dr. Zimmerman).

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Impressed by (1) the frequency with which eyes having this preventable, and often curable, condition are enucleated, and (2) the existing confusion concerning the relation of glaucoma to pathologic changes in the lens, we decided to report our observations based on a study of 138 such cases in the Registry of Ophthalmic Pathology of the Armed Forces Institute of Pathology.

Why were so many of these eyes enucleated? Was the problem one of diagnosis or of management? Were these difficulties based on lack of understanding of the basic pathology and pathogenesis of this type of acute glaucoma?

MATERIALS AND METHODS

From the files of the Registry of Ophthalmic Pathology, all cases coded as phacogenic glaucoma and glaucoma due to hypermature cataract and all other cases of hypermature cataract, as well, were reviewed for possible inclusion in the study. Only those cases were accepted in which the pathologic process was clear-cut. All cases had at least the following features: (1) definite clinical or pathologic evidence of glaucoma; (2) the presence of a hypermature cataract (spontaneous or traumatic), as evidenced by fluid cortex seen microscopically, and (3) the presence outside the lens capsule of characteristic large phagocytes containing ingested lens substance.

The material available for study included microscopic slides of every eye. Clinical features were obtained from Registry records, and particular attention was directed to age, sex, race of the patient, clinical diagnosis, symptoms, condition of the other eye, duration of symptoms before enucleation, and treatment.

Statements of the clinician are reported as he gave them. The relative frequency of the symptoms and signs as given is not to be considered a statistically accurate indication of their incidence. Instead, they are the clinical manifestations that impressed the clinician enough to move him to record them on the

Registry form. Nevertheless, the list of clinical findings helps to indicate the difficulties faced by the clinician.

CLINICAL ASPECTS

Age.—Acute glaucoma of this type is a disease of advancing age. Although the ages ranged from 29 to 94 years, a majority of the patients in this series were over 70. Only four were less than 49, and each of these patients had a retained intraocular foreign body. The incidence rises sharply at age 65 (Fig. 1).

Sex and Race.—There were 78 men and 52 women; the sex of 8 was not stated. Eight patients were Negroes; 123 were white; the race of 7 was not recorded.

Symptoms and Signs.—A distinction must be made between two groups of symptoms in these patients. Since the type of glaucoma under consideration is a complication of hypermature cataract, all of these patients had seriously impaired vision prior to the onset of acute glaucoma. Their recorded visual acuity varied from no light perception to ability to distinguish hand movements, but many were simply said to be "blind."

The second group of symptoms is related to the onset of glaucoma, which in most cases was sudden and varied little from the well-known picture of acute "congestive glaucoma"—ocular and periorbital pain, headache of varying severity, nausea, vomiting, and prostration. In the younger patients the onset was more insidious. The eyes were usually congested and showed corneal edema, aqueous flare and cells, fixed, often dilated, pupil, and grossly deep anterior chamber. The signs, however, were not always constant, and small pupils, shallow chambers, subluxation of the lens, and hyphema were also recorded. The reported intraocular tensions varied from 30 to over 100 mm. Symptoms of acute glaucoma were present for less than three weeks prior to enucleation in 71% of the 109 cases in which these data were available; enucleation was performed during the first week in 37% (Table 1).

TABLE 1.—*Duration of Symptoms of Glaucoma Prior to Enucleation*

Duration	No. of Cases
0-48 hr.	8
48 hr.-1 wk.	32
1-3 wk.	38
3-5 wk.	4
5-8 wk.	12
8-16 wk.	4
4 mo.-1 yr.	4
1-4 yr.	7
Total	109

Clinical Course.—The typical case concerns a patient beyond the age of 65. A cataract is present in one eye, but the opposite eye has good vision, though aphakic in half the cases. Then there is the sudden onset of mild to severe pain in the cataractous eye. Examination reveals marked congestion, corneal edema, an aqueous flare and cells, and sometimes a denser accumulation of exudate in the anterior chamber. Glistening particles may also be observed. The intraocular tension is elevated. After a brief trial of medical therapy, enucleation is performed because of a "blind," painful eye.

Condition of Other Eye.—Information about the opposite eye was available in 89 cases. On the basis of this information the cases could be divided into three groups. Group I, the largest, consisting of 39 cases, included those patients whose opposite eye was aphakic but with good vision. These patients were uniformly elderly, and in them the onset of glaucoma was almost always violent. The sudden development of glaucoma in the cataractous eye of an old person

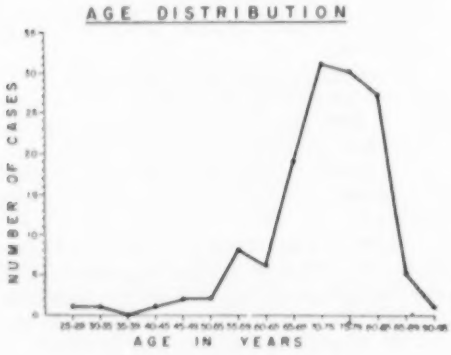


Fig. 1.—Age distribution.

TABLE 2.—*Clinical Diagnoses Recorded by Contributors*

Diagnoses	No. of Cases
Absolute glaucoma	36
Acute glaucoma secondary to hypermature cataract	21
Secondary glaucoma	21
Acute congestive glaucoma	17
Iritis, iridocyclitis, or uveitis	14
Intraocular tumor	11
Glaucoma	10
Subluxation or dislocation of lens	8
Hemorrhagic glaucoma	5
Intraocular foreign body	4
Chronic glaucoma, acute exacerbation	2
Inflammatory glaucoma	2
Endophthalmitis	2
Panophthalmitis	2
Infectious glaucoma	1
Iris bombé	1
Keratitis	1
Chronic congestive glaucoma	1
Hypopyon	1
Glaucoma following swollen cataract	1
Eye blind and diseased	1
Total	163*

* More than one diagnosis was listed in some cases.

whose fellow eye is aphakic constitutes a clinical syndrome which should be considered phacolytic glaucoma until proved otherwise. Group II, the second largest, included patients with monocular cataract and good vision in the opposite eye. All of the traumatic cataracts were in this group, although the majority of the cataracts were of uncertain origin. Many probably represented cases of senile cataract in which the process advanced much more rapidly on one side than on the other. Group III consisted of 10 patients whose opposite eye had impaired vision, due to incipient cataract in 7 and mature cataract in 2, and of unknown cause in 1.

Differential Diagnosis.—In 21 cases (15%) the correct diagnosis of glaucoma secondary to hypermature cataract was made by the attending ophthalmologist. Other clinical diagnoses made are listed in their order of frequency in Table 2. "Absolute glaucoma" was the diagnosis made most frequently. The violent onset of symptoms and the congested eye account for the frequent diagnosis of acute congestive glaucoma and also for the fact that iridectomy was the surgical procedure most commonly employed. The diagnosis of uveitis is explained by the presence of aqueous flare and cells. Inability to visualize the fundus, the lack of response to therapy, and the consideration that tumor may have caused the cataract are factors probably responsible for the frequent diagnosis of intraocular tumor. A miscellany of conditions is included in the remaining 32 diagnoses listed in Table 2.

Treatment.—Treatment included the whole range of standard therapeutic measures for control of glaucoma. Miotics were used most frequently—in 40 cases. Surgical procedures of various types were performed in 13 cases. Mydriatics, cortisone, and acetazoleamide (Diamox) were used in a few cases.

PATHOLOGY AND PATHOGENESIS

The lenticular degeneration essential for the development of this type of glaucoma is liquefaction of the cortex (Fig. 2). Although it may occur focally, liquefaction was usually extensive, and in 86 (62%) the entire cortex seemed to be involved † (Fig. 3). Degenerative changes were usually observed in the lens epithelium and capsule. However, the statement often made that normal lens epithelium is not found in hypermature cata-

† All such statements are based on examination of one or several microslides, but in no case were serial sections of the entire lens or eye available for this study.

Fig. 2.—Advanced degeneration of lens cortex, much of which is liquefied (*l*). Only the peripheral cortical fibers (*c*) near the equator remain relatively intact. The sclerotic nucleus (*n*) is seen at the lower right. A. F. I. P. Accession No. 187691; reduced ⅔ from mag. × 50.

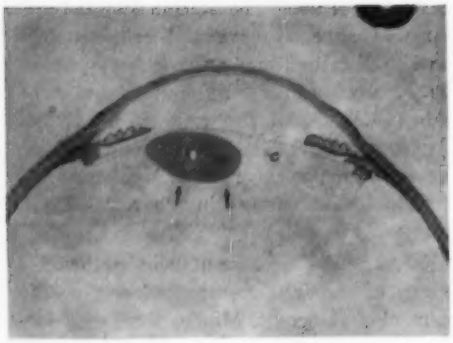


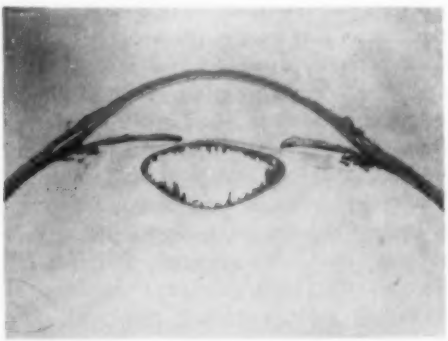
Fig. 3.—Phacolysis has involved the entire lens cortex (*c*). Only the sclerotic nucleus (*n*) remains intact within the lens capsule, which is markedly attenuated posteriorly (arrows). The anterior chamber is deep, and there are no synechiae. A. F. I. P. Accession No. 57055; reduced ⅔ from mag. × 7.

Fig. 5.—The entire lens cortex (*c*) has become liquefied, and there is advanced degeneration of the lens capsule, which has only recently ruptured posteriorly (arrow). Histiocytes have collected over the outer surface of the lens and are especially numerous where capsular degeneration is most marked. Only an occasional histiocyte is observed within the lens capsule. A. F. I. P. Accession No. 313464; reduced ⅔ from mag. × 45.

racts was proved false, for in 18 (13%) of these lenses segments of normal capsule and normal epithelium were found (Fig. 4). The most advanced alterations were observed posteriorly, where often the capsule was attenuated markedly. In 22 (16%) of the lenses there was evidence that the capsule had ruptured (Fig. 5). In the others liquefied cortical material had apparently escaped through the unruptured capsule and could be seen over the surface of the lens and in the anterior chamber. With the escape of liquefied cortex, the lens shrinks and may assume an odd contour. When the loss of cortex is symmetrical and the nucleus remains in a central position, the anterior and posterior capsules near the equator may be closely approximated (Fig. 6). Rarely is there significant lysis of the nucleus, but in one eye the entire lens substance had escaped and only the empty capsule remained (Fig. 7). The nuclear alteration more frequently observed is dislocation. Floating in the Morgagnian fluid, the sclerotic nucleus may settle to the inferior part of the lens, where it is seen in various eccentric positions. In 25 lenses (18%) doubly refractile crystalline

Fig. 4.—A segment of well-preserved epithelium and capsule in a lens in which there was much liquefaction of lens cortex (*c*). A. F. I. P. Accession No. 187691; reduced ⅔ from mag. × 155.

Fig. 6.—Complete cortical phacolysis and escape of liquefied lens substance have led to marked shrinkage and approximation of the anterior and posterior capsules near the equator. Absence of nuclear material is an artifact. The deep anterior chamber and lack of synechiae are characteristic features. A. F. I. P. Accession No. 612178; reduced ⅔ from mag. × 7.



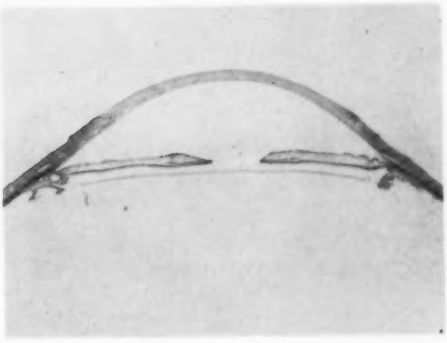


Fig. 7.—Phacolysis has involved the entire lens substance, and the liquefied material has escaped through the apparently unbroken capsule, which remains in its normal position. Again to be observed are the characteristically deep anterior chamber and absence of synechiae. A. F. I. P. Accession No. 235329; reduced ⅔ from mag. × 8.

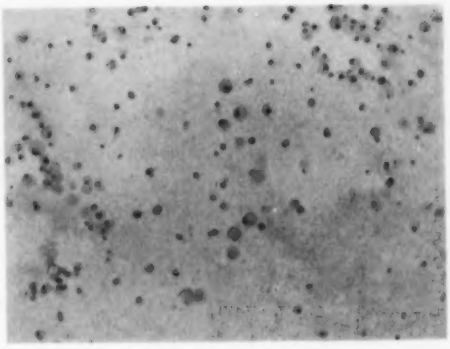


Fig. 9.—Large numbers of histiocytes in a sea of liquefied lens material as they are observed in the anterior chamber. The smaller cells represent mononuclear phagocytes that have not yet gorged themselves with liquefied lens substance. Characteristically, other types of inflammatory cells are few or absent. A. F. I. P. Accession No. 305232; reduced ⅔ from mag. × 165.

deposits, morphologically identical with the calcium oxalate crystals found in old detached retinas, were observed in the sclerotic nucleus.

Liquefaction of the lens cortex, a process which may be called phacolysis, is said by Krause [14] to be mediated by two enzymes—protease A and protease B. Through their activity large molecules of lens protein are disintegrated to small molecules, which apparently diffuse through the often intact, though perhaps more permeable, lens capsule. The presence of this lysed cortical material in the posterior and anterior chambers evokes a histiocytic response. Large mononuclear phagocytes collect over the lens surface (Fig. 8) but are not observed within the lens unless the capsule is broken (Fig. 5). These macrophages have a very characteristic appearance once they have become engorged with liquefied lens substance (Fig. 9). They swell up and become rounded. Their cytoplasm is distended with finely granular, pale-staining, slightly acidophilic particles of lens material, almost identical with that still remaining within the lens. Carried by aqueous flow, these macrophages become relatively more concentrated in the iridocorneal

Fig. 8.—Collection of histiocytes embedded in a matrix of escaped lens fluid adheres to the outer surfaces of the lens capsule. No cells are seen within the lens capsule. A. F. I. P. Accession No. 197063; reduced ⅔ from mag. × 70.

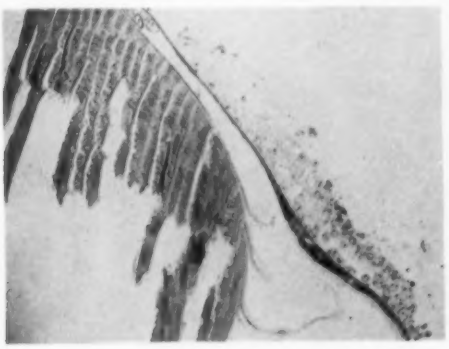
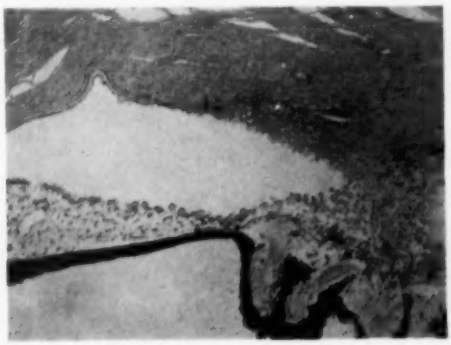


Fig. 10. Histiocytes become concentrated in the anterior chamber angles, on the anterior surface of the iris, and over the trabecula. A. F. I. P. Accession No. 609920; reduced ⅔ from mag. × 75.



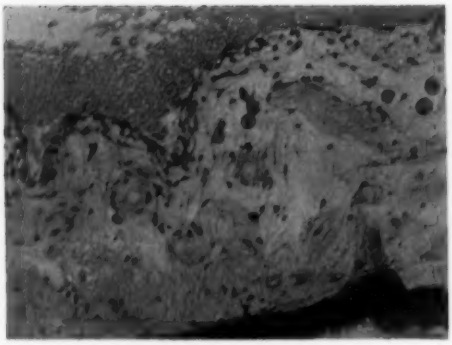


Fig. 11.—Typical macrophages laden with liquefied lens material are seen in the iris crypts and within the iris stroma. The anterior chamber (*a c*) is filled with escaped lens substance, which is completely liquefied. A. F. I. P. Accession No. 643703; reduced ⅔ from mag. × 165.

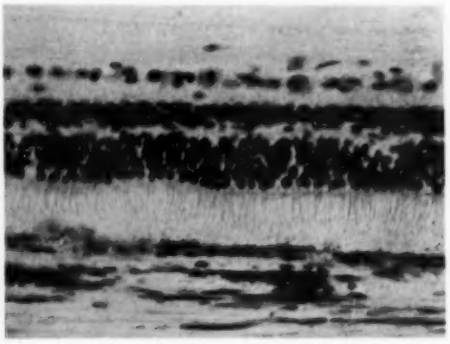
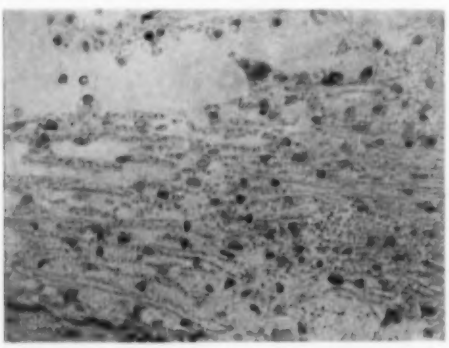


Fig. 13.—(*A*) preservation of retinal architecture with no evident atrophy of ganglion cells or nerve fibers reflects the recent onset of glaucoma and indicates the good prognosis if cataract extraction is performed. A. F. I. P. Accession No. 207298; reduced ⅔ from mag. × 410.

angles than elsewhere in the anterior chamber (Fig. 10). They fill the iris crypts and may enter the iris stroma (Fig. 11). More important, though, is their effect on the filtration apparatus. The intertrabecular spaces and the surface of the trabecula become filled with these cells and with free lens material present in the aqueous (Fig. 12). These macrophages apparently are not "sticky," for they do not agglutinate or form keratic precipitates, an important differential point clinically as well as pathologically.

In 108 (78%) of these cases pathologic study revealed a typical picture—hypermature cataract, liquefied lens substance in the posterior and anterior chambers, and the characteristic histiocytic response. No other cells were present in significant numbers, and there was no other demonstrable cause for glaucoma. Neither anterior nor posterior synechiae had formed, and the anterior chamber characteristically was deep (Figs. 3, 6, and 7). This observation is of special importance to the pathologist, and it should be of value to the clinician also. Most eyes removed because of glaucoma of

Fig. 12.—Histiocytes may be seen in the intertrabecular spaces, as well as on the surface of the trabecula. In addition, an abundance of liquefied lens material fills and obstructs the intertrabecular spaces. The scleral spur (*s p*) is at the lower right, while the anterior chamber angle (*a c*) is at the upper left. A. F. I. P. Accession No. 168726; reduced ⅔ from mag. × 385.



(*B*) As is typical of phacolytic glaucoma of recent onset, the disc is not cupped, and there is no significant depression of the lamina cribrosa. A. F. I. P. Accession No. 232645; reduced ⅔ from × 19.

other types have synechiae and a shallow chamber by the time the eye is enucleated. The cases in the present series represent the most important exception to this rule. As the Irvines ‡ demonstrated, the fluid in the anterior chamber lacks fibrinogen, and this, together with the short duration, probably accounts for the absence of synchiae in phacolytic glaucoma.

In 30 cases other alterations were present. For reasons we do not understand, there was hemorrhage into the anterior chamber in 10 cases, and in another 3 cases a considerable number of polymorphonuclear leucocytes was present. Although in typical cases the inflammatory reaction in the uveal tract was minimal, iritis or iridocyclitis of nonspecific character was observed in four cases. In eight cases the cataract was traumatic, and in some of these eyes there were additional pathologic changes, related to the original injury. Nevertheless, glaucoma in these eyes was also regarded as secondary to escape of hypermature lens material. In six eyes there was evidence of glaucoma surgery.

A most important characteristic of these cases was the relatively good state of preservation of the retina, disc, and optic nerve (Fig. 13). In fact, in 21 cases (15%) it was impossible to make a dogmatic histopathologic diagnosis of glaucoma. These eyes showed no loss of ganglion cells, no atrophy of the nerve fiber layer, no cupping of the disc, no depression of the lamina cribrosa, and no atrophy of the nerve. In an additional 80 cases (58%) glaucomatous changes, though present, were considered mild. This, of course, reflects the short duration of glaucoma in most of these patients and explains the good visual results that have been reported by the Irvines and others following removal of the hypermature lens and irrigation of the anterior chamber.

COMMENT

Glaucoma of the type with which we have been concerned in the present study is a pathologic entity, clinically recognizable, readily preventable, and often curable by cataract extraction.

We propose the name "phacolytic glaucoma" to describe this malady. It is a concise term denoting the basic pathologic process, namely, dissolution of the lens. Some of the other terms previously used to describe it are (1) lens-induced glaucoma, (2) phacogenetic glaucoma, (3) phacogenic glaucoma (4) glaucoma associated with Morgagnian cataract, (5) glaucoma associated with hypermature cataract, and (6) glaucoma due to spontaneous rupture of hypermature lens.

Lens-induced glaucoma, phacogenetic glaucoma, and phacogenic glaucoma are objectionable terms in that they provide no clue as to the way in which glaucoma is related to pathologic changes in the lens. These names might be equally applicable to the glaucoma seen in association with exfoliation of the lens capsule, spherophakia, endophthalmitis due to lens substance, and intumescent cataract, all of which may be confused with phacolytic glaucoma. The basic mechanism in each of these is different. The mechanism of glaucoma due to exfoliation of lens capsule, like that due to liquefied lens cortex, is essentially obstruction of the intertrabecular spaces by particulate matter. In exfoliation it is the flakes of lens capsule that produce the obstruction, while in phacolytic glaucoma it is a combination of phagocytic cells and escaped Morgagnian fluid. In spherophakia and intumescent cataract glaucoma is due to pupillary block. Glaucoma associated with lens-induced endophthalmitis may be due to anterior or posterior synechiae or to obstruction of the chamber angles by inflammatory exudate. In the latter situation, the pathologic changes differ from those of phacolytic glaucoma in the character of the inflammatory exudate. That in lens-induced endophthalmitis consists of large numbers of lymphocytes, plasma cells, epithelioid cells, and polymorphonuclear leucocytes, while the typical reaction in phacolytic glaucoma is a bland outpouring of macrophages.

In our experience, the "chemical irritation" theory of glaucoma associated with hypermature cataract confuses more than it enlightens.

‡ References 7 to 9.

All too often this form is regarded as a type of hypersecretion glaucoma, the so-called toxic effect being on the ciliary body. We do not deny that there is chemical irritation, but we believe that its main deleterious effect is the histiocytic response and consequent plugging of trabecular spaces by macrophages.

Likewise, we doubt that A. Knapp's [15] suggestion of irritation of the ciliary body by the bouncing lens nucleus of a Morgagnian cataract accounts for many cases of phacogenic glaucoma. Also, we believe that capsular rupture is not an essential factor and that evidence for its occurrence is lacking in many of the reported cases.

Comprehension of the pathologic basis for these different types of lens-induced glaucoma is not without therapeutic significance. In phacolytic glaucoma, we believe that after delivery of the lens, the anterior chamber should be irrigated in an effort to remove as much Morgagnian fluid and as many macrophages as possible.

Three of the outstanding clinical features of this group of patients were (1) the sudden onset of glaucoma in an eye that had been blind from cataract for many years, (2) the patient's advanced age, and (3) the almost constant observation of good vision and normal tension in the opposite eye. Pathologically there was evidence that the cataracts had been liquefied for a long time; yet the retinas and optic nerves in a significant number were well preserved. This observation is support for the belief that glaucoma was of recent origin.

How, then, can the variable, though usually sudden, onset of glaucoma be explained? At least three factors seem to be at work: 1. The rate at which lens fluid escapes into the anterior chamber. The sudden onset of symptoms is not necessarily indicative of rupture of the lens capsule. In the majority of our cases pathologic evidence of rupture was lacking. Probably, escape of lens fluid depends upon (*a*) the degree of hydrolysis of lens protein into smaller, diffusible molecules, and (*b*) degeneration of lens capsule, so that it becomes more permeable. 2. The age of the patient. In 1937, Howard Knapp [3] postulated that the drainage facility of the elderly patient's eye was less efficient than that of more youthful persons. Other workers § have observed that old patients do not handle escaped lens substance as well as young ones. In our younger patients the course was insidious or intermittent, in contrast to the acute onset in older patients. 3. The presence of coexisting eye disease, such as chronic simple glaucoma, siderosis bulbi, or chronic iridocyclitis. This factor was relatively less important than (1) and (2), but probably the reason this type of glaucoma develops in some young patients is that their filtration mechanisms have been rendered less efficient by other eye disease. It is significant in this regard that the four patients in our series below the age of 50 had had intraocular foreign bodies and two had well-developed siderosis bulbi.

Even though the correct diagnosis was made in 21 cases, the physician did not elect to extract the lens which is the seat of the trouble. This, then, is a plea for cataract extraction. Although the eye is suffering from an acute, and often violent, disturbance and the patient says that he has been "blind" in this eye for many years and shows faulty light projection, cure and salvable vision may be the reward if cataract extraction and anterior chamber irrigation are done.

SUMMARY

A study of 138 eyes enucleated because of glaucoma associated with hypermature cataract establishes this type of glaucoma as a clear-cut pathologic entity. Liquefaction of the lens cortex and degeneration of the lens capsule and epithelium permit escape of the lens liquid into the anterior chamber. The lysed cortical material evokes a histiocytic response. Macrophages, swollen with phagocytosed lens material, block the filtration apparatus, to produce acute glaucoma. We propose the name "phacolytic glaucoma" for

§ Maumenee, A. E.: Personal communication to the authors. Fine, M.: Personal communication to the authors.

this condition, for the reason that it is descriptive of the basic pathology.

Review of the case histories reveals characteristic clinical features. The acute onset of glaucoma in the cataractous eye of an old person with good vision in the other eye, particularly if the other eye is aphakic, should lead to serious consideration of phacolytic glaucoma. A deep anterior chamber, lack of synechiae, and lack of keratic precipitates in the presence of aqueous flare and cells lend further support to this diagnosis.

Comprehension of the pathology and pathogenesis of phacolytic glaucoma should lead to the correct treatment of this surgical emergency, namely, cataract extraction and irrigation of the anterior chamber. Moreover, this type of glaucoma is completely preventable by cataract extraction before the development of hypermaturity.

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Prednisone (Meticorten) and Prednisolone (Meticortelone) in Ophthalmology

Experimental and Clinical Studies

COL. J. H. KING (MC), U. S. Army
and
CAPT. JOHN R. WEIMER (MC), U. S. A. F.

The advent of the adrenal corticosteroids a few years ago marked a new era in ocular therapeutics. Before the values of cortisone had been finally assessed by researchers, this hormone was somewhat overshadowed by the introduction of its more potent relative, hydrocortisone. The brief passage of time has not allowed complete clarification of their roles in ophthalmology; however, certain facts have become well established. The value of adrenal steroids as potent therapeutic agents in ocular inflammation and allergic states is accepted. The indications, contraindications, dosages, routes of administration, and principal physiologic effects have been well outlined by many authors from clinical and experimental studies.

The horizon of this new era of adrenal steroids has been extended by the recent synthesis of two more potent and less toxic corticosteroids, prednisone * (Meticorten) and prednisolone † (Meticortelone). It is the purpose of this preliminary report to introduce these new substances to the field of ophthalmology.

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Meticorten and Meticortelone for this study were supplied by the Division of Clinical Research, Schering Corporation, Bloomfield, N. J.

From the Ophthalmology Service, Walter Reed Army Hospital, and the Ocular Research Unit, Walter Reed Army Medical Center, Washington 12, D. C.

* Prednisone, formerly known by the generic name metacortandracin.

† Prednisolone, formerly known by the generic name metacortandralone.

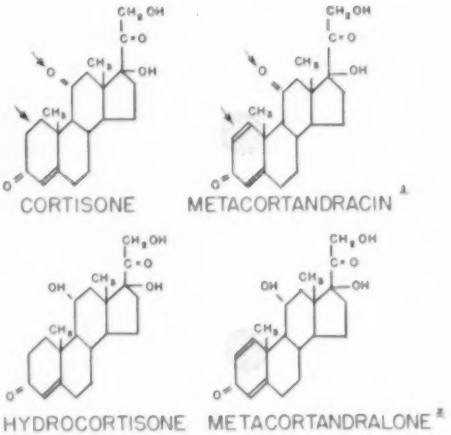

Fig. 1.—Chemical structure of four adrenal corticosteroids, *1*, metacortandracin, now known as prednisone (Meticorten). *2*, metacortandralone, now known as prednisolone (Meticortelone).

PAST STUDIES

These synthetic corticosteroids differ from cortisone and hydrocortisone in that they possess an additional double bond in the A ring conjugated to the ketone. Prednisolone differs from prednisone in the same way that hydrocortisone differs from cortisone, that is, by possessing a hydroxyl group in the 11 position, rather than a carbonyl group (Fig. 1).

Biological and clinical studies show that these substances are two to four times as active as cortisone and hydrocortisone. At the same time, the undesirable side-effects and toxic reactions are less than those experienced with other steroids. There is less potassium excretion and very little sodium retention in therapeutic dosage.

Preliminary clinical reports indicate that these drugs possess potent anti-inflammatory and antiallergic properties. They have been

found to be effective antirheumatic agents in the short-term therapy of rheumatoid arthritis.‡ Pemphigus vulgaris[8] and extensive eczematous dermatitis § have been successfully treated with prednisone. Marked improvement has been reported in bronchial asthma and pulmonary emphysema and fibrosis by using the same agent.‖ Not only was it considered more effective and safer, but therapeutic response was greater than that obtained with cortisone and hydrocortisone. Intractable bronchial asthma, or status asthmaticus, has been brought under swift control with prednisone.[2]

PRESENT STUDIES

Animal experimentation and clinical studies were performed to verify the presumption that prednisone and prednisolone were as effective as or more potent than cortisone and hydrocortisone in the treatment of ocular inflammation known to respond to the latter hormones.

EXPERIMENTAL OBSERVATIONS

Experiment 1

Fifteen young rabbits were sensitized to horse serum by an intravenous injection of 10 cc. of filtered sterile unpreserved horse serum.¶ All animals were examined and found free of spontaneous uveitis. During the waiting period three of the rabbits died of intercurrent cage infection. On the 14th day of the experiment the 12 remaining animals were given a shocking dose of 0.1 cc. of the same horse serum intravitreously. They were then divided into three groups of four animals each. Group I (the control group) was given daily injections of sterile saline intramuscularly. Group II received daily intramuscular injections, 25 mg. per kilogram of body weight, of sterile cortisone suspension (Merck). Group III was administered daily intramuscular injections of 10 mg. per kilogram of body weight of sterile prednisone. It is to be noted that Group II received two and one-half times the strength of medication of Group III.

The animals were examined daily and the reactions divided into three types: vitreous haze, anterior-chamber flare, and inflammatory exudate on the anterior surface of the lens. Each of these responses was graded 1+ to 4+, thus giving a total reaction of 12+ in an animal which demonstrated a dense diffuse vitreous haze, a marked anterior-chamber flare and cells, and complete ringing of the lens with inflammatory exudate at the iris border. On this basis, the reactions of the groups were graded daily and the results shown in Figure 2.

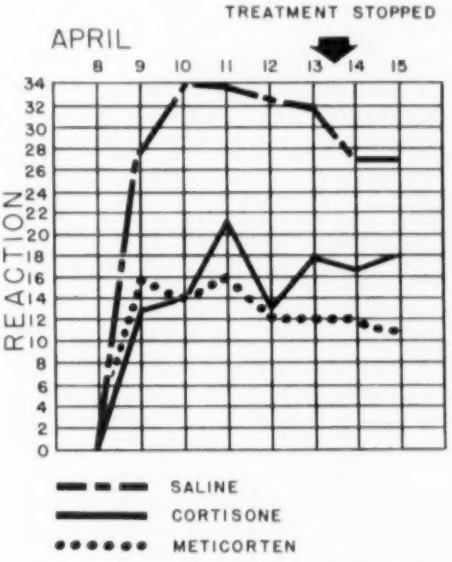

Fig. 2.—Graph of results in animal experiments.

Results.—The treatment with either cortisone or prednisone was effective in limiting the inflammatory response. Cortisone was slightly less effective in doses two and one-half times that of prednisone. The results of the comparison of cortisone-treated animals with the control animals were similar to the findings of Biegel.[5]

Experiment 2

On the 20th day of Experiment 1, two days after treatment had been discontinued, most of the inflammatory responses had abated. Animals who continued to exhibit any marked inflammation were injected subconjunctivally with 0.4 cc. of 0.5% prednisone or 0.4 cc. of 0.5% hydrocortisone. If the animals were in the prednisone-treated group in Experiment 1, they received sub-

‡ References 1 and 3.

§ Flood, J. M.: Personal communication to the authors.

‖ References 2 and 4.

¶ Courtesy of Wyeth Laboratories, Inc., Philadelphia.

conjunctival prednisone, and if they were in the cortisone-treated group in Experiment 1, they received subconjunctival hydrocortisone. In all, two animals were injected with hydrocortisone and three with prednisone. No untoward effects of the injections were noted in either group, but no clearance of the reaction, which involved the entire uveal tract, resulted at this dosage level.

Experiment 3

The same animals injected in Experiment 2 were further observed, and, as no changes in their eye conditions were noted in 72 hours, they were injected subconjunctivally with 0.1 cc. of prednisone, 10 mg. per cubic centimeter, and 0.1 cc. of hydrocortisone, 25 mg. per cubic centimeter. Again, no ill-effects resulted, but a definite subsidence of the ocular inflammatory response occurred in both groups.

It was interesting to note that three of the steroid-protected animals developed an ocular infection from the intravitreous injection, while none of the control groups showed evidence of infection. These occurrences may have been a failure of the medication to limit the inflammatory reaction. Owing to the small number of animals in the series, however, they were retained for live study, and no attempt to prove infection histologically was made. One of these three animals eventually developed a frank purulent panophthalmitis.

Both Biegel [5] and Woods and Friedenwald [6] have shown the pathological reactions upon which our experiments were based.

Clinical Observations

In general, the accepted indications for corticosteroid therapy were used in selecting our clinical material.[7] Fifty-four cases were included, with a total of 67 eyes treated by the medications in these studies.

Routes of administration included oral, topical, and subconjunctival. The only oral and subconjunctival medication available was prednisone. For topical use prednisone and prednisolone were available in both the acetate and the alcohol form. These were used as ointments and as suspensions. The ointments were all of 0.5% strength with the exception of prednisone alcohol, which was also used in 0.1% strength. The suspensions were prepared in benzalkonium (Zephiran) chloride 1:10,000 and were used in strengths of 0.1%, 0.25%, and 0.5%. The subconjunctival preparation was a sterile prednisone suspension with a strength of 10 mg. of prednisone per cubic centimeter.

In order to facilitate charting, the dosage schedule followed will be noted only if it differs from the schedule given here. For oral medication, 30 mg. a day in divided doses of 10 mg. three times a day, was administered until a therapeutic effect was achieved or an arbitrary seven-day limit was reached. When one of these two conditions was fulfilled, the dosage was decreased 5 mg. daily until a maintenance dose was attained, and this was considered to be 5 mg. daily unless otherwise noted.

The topical application was given every two hours unless otherwise noted.

The cases were divided into five groups as follows:

1. Iridocyclitis
2. Diffuse uveitis
3. Postoperative inflammation (not complications)
4. Marginal corneal ulcers
5. Miscellaneous conditions

Iridocyclitis.—In this series we have treated 13 patients with iridocyclitis, and the results of therapy in the 16 eyes involved are recorded in Table 1. Owing to limitations of space, no attempt is made to relate the detailed histories in all of these instances, but several cases will demonstrate certain principles of treatment. Topical medication was found to be effective within an average of 4 to 5 days, and in some cases a marked response was noted within 12 hours.

In this Table and in the following Tables results were graded as slight, moderate, and marked. The response was judged by diminution and rapidity of diminution of cells and flare in the anterior chamber, by relief of ciliary flush if present, and by changes in

visual acuity. A question mark preceding the response indicates that this may or may not have been the direct result of the corticosteroid therapy.

In all cases of nongranulomatous iridocyclitis, i. e., primary or secondary to trauma, there was a moderate to marked response to the topical corticosteroids with the exception of one eye, which contained a retained foreign body (Case 10, Table 1).

In the two patients with Boeck's sarcoid the only eye involvement noted was an anterior and a slight diffuse posterior inflammatory reaction with no discrete local lesion in evidence. It was clearly demonstrated that the corticosteroid is not a cure in these cases but does limit the inflammatory response and minimizes the formation of synechiae and other complications.

In these two instances (Cases 11 and 14, Table 1) we were able to evaluate several of the drugs and their relative potency. Cases 11 and 12 were found to respond well to prednisone acetate ointment and hydrocortisone alcohol ointment, both 0.5%, but improvement was most marked with prednisolone alcohol 0.5% ointment. Prednisone alcohol ointments in strengths of 0.1% and 0.5% were found to be equally effective, but in both strengths the ointment was less effective than the prednisone acetate and prednisolone alcohol forms in the same strengths.

All treatment was discontinued on two occasions in the two cases (11 and 12), and within one to two days a gradual build-up of inflammatory reaction and increased synechial formation occurred. No systemic therapy was attempted, inasmuch as both patients had generalized sarcoidosis, and it was the opinion of the medical consultant that such therapy might be dangerous.

TABLE 1.—*Iridocyclitis*

Case	Diagnosis	Treatment	Days of Disease	Days to Improvement	Days to Remission	Response
1 J. B.	Recurrent, O. D. (nongranulomatous)	Prednisone acetate 0.5% every 2 hr.; oxymycin h. s. & atropine	5	2	..	Moderate
		Prednisone 20 mg. daily	20	5	..	Marked
2 A. A.	Acute, O. D. (nongranulomatous)	Prednisone acetate 0.5%	52	(?)	3	Moderate
3 D. A. C.	Acute, O. S. (nongranulomatous)	Prednisolone alcohol 0.5%	1	2	2	Marked
4 L. G. M.	Acute (nongranulomatous)	Prednisolone alcohol 0.5%	14	1	3	Marked
5 H. T.	Acute (nongranulomatous)	Prednisolone alcohol 0.5%	14	1	3	Marked
6 E. D. H.	Associated with rheumatoid arthritis, 8 yr. duration	Prednisone acetate 0.5%	1	1	..	Marked
7 T. P.	Associated with rheumatoid arthritis, O. S.	Prednisolone alcohol 0.5% q. i. d.	1	3	5	Moderate
8 G. J. C.	Secondary to corneal foreign body	Prednisolone alcohol 0.5% every 2 hr.	2	1	1	Marked
9 W. G. S.	Secondary to trauma, O. S.	Prednisone 30 mg. daily & hydrocortisone 0.5% topically	27	4	4	Moderate
10 W. G. S.	Secondary to trauma	Prednisone 30 mg. daily; hydrocortisone 0.5% topically	27	7	..	Slight *
11 S. B.	Anterior sarcoidosis, O. D.	Corticosteroids every 2 hr. O. U. topically only	160	10	..	Moderate
12 S. B.	Anterior sarcoidosis, O. S.	Same	160	4	..	Moderate
13 H. H.	Anterior sarcoidosis, O. D.	Placebo and corticosteroids	180	5-14	..	Slight
14 H. H.	Anterior sarcoidosis, O. S.	Placebo and corticosteroids	180	5-14	..	Slight
15 E. B.	Associated with heterochromia	Prednisolone alcohol	(?) 4 yr.	1	..	Marked
16 H. J. T.	Granulomatous, O. S.	Hydrocortisone 0.5%	6	6	..	Slight
		Prednisolone acetate solution 0.5% and prednisone	40	2	.	Marked

* See text.

Most of the cases of iridocyclitis reacted so rapidly that only limited information could be obtained regarding the comparative effectiveness of the various preparations. On the whole, however, the prednisolone preparations and the alcohol forms of each drug were more effective.

Two patients (Cases 10 and 16, Table 1) did not respond to topical medication but did react to oral prednisone.

Diffuse Uveitis.—Five cases of diffuse uveitis, with a total of seven eyes, were treated with various corticosteroids. The results are shown in Table 2.

Fig. 3.—Ciliary flush.

TABLE 2.—*Diffuse Uveitis*

	Case	Diagnosis	Treatment	Days of Disease	Days to Improvement	Days to Remission	Response
1	C. E. H.	Anterior and posterior secondary to anterior-placed choroidal lesion O. D.	Prednisone acetate 0.5%	14	5	..	Slight
			Prednisone	19	4	..	Marked
2	W. L. H.	Associated with macular involvement, O. D. only	Prednisone	47	..	8	(?) Slight *
3	J. D. H.	? Toxoplasmosis recurrent, O. D. only	Prednisone	6	3	..	Marked †
4	R. J.	Diffuse chorioretinitis, O. D.	Prednisolone alcohol 0.5%	48	..	..	None
			Prednisone	52	2	8	Marked
5	R. J.	Diffuse chorioretinitis, O. S.	Prednisolone alcohol 0.5%	48	..	..	None
			Prednisone	52	2	8	Marked
6	W. R. M.	Diffuse chorioretinitis, O. D.	Prednisone alcohol 0.1%; hydrocortisone, 0.5% ointment; prednisone alcohol 0.5%; prednisolone acetate 0.5% and plain ointment base placebo	69	..	..	No response
			Prednisone	92	2	..	Marked
7	W. R. M.	Diffuse chorioretinitis, O. S.	Prednisone	92	1	3	Marked

* Had been clearing.
† See text.

Topical applications were employed in three of these cases, with only slight effect in one eye and no response in the other four eyes. However, in four of the five patients and in six of the seven eyes, the administration of oral prednisone brought about a dramatic decrease in the objective inflammatory signs and marked subjective improvement in vision, due to clearing of the ocular media.

A large amount of data on dosage levels was obtained by studying the therapeutic effects on one patient (Cases 4 and 5, Table 2). Marked diminution of the inflammatory response was noted within 12 to 48 hours after institution of therapy upon four different occasions, and there was a marked recurrence of the inflammation within 24 hours after discontinuance of the medication. These recurrences were evidenced twice by a marked ciliary flush (Fig. 3). A study of this case also permitted an evaluation of the effective maintenance dose. The first recurrence took place immediately upon discontinuing the initial daily dose of 30 mg. of prednisone. The medication was again started at a dosage of 30 mg. daily and continued until the ocular media cleared, and then was reduced 5 mg. daily to 10 mg. a day. It was maintained at this level for 16 days without any cells or flare appearing in either anterior chamber. Within 24 hours after discontinuing the medication from this level, the ciliary flush

(Fig. 3) reappeared, and cells and flare were present in both anterior chambers. This inflammation was again controlled with an initial dose of 30 mg. daily, and upon subsidence of the inflammatory response the dosage level was reduced again 5 mg. daily to a dose of 5 mg. daily, which maintained the suppression of the inflammatory response. Upon discontinuance of this dosage level after 36 days, a marked inflammatory build-up again occurred, and this was controlled easily with a return to the initial prednisone therapy. It is evident that at least in some cases 5 mg. daily may be a very effective maintenance dose. We have seen no instance where an inflammatory response which was effectively controlled with prednisone recurred at this dosage level.

The rationale of therapy in these instances, as with other steroid medications, is the prevention of the secondary detrimental effects of chronic inflammatory reaction. The actual etiology is unknown in many instances, and it is hoped that the eye can be preserved until the natural body forces can mobilize against the offender. In one case (1, Table 2) the reduction of the inflammatory reaction in no way appeared to limit the production of pigment and the apparent healing of the chorioretinal lesion. In another case (4, Table 2) the scotomata from chorioretinal lesions continued to enlarge and the vision to diminish despite the absence of external inflammatory signs and the clarity of the media. In the same patient's other eye (5, Table 2) such was not the case, as the scotomata decreased in size and the vision returned to 20/30.

Postoperative Inflammation.—Hydrocortisone has been used to reduce inflammation following certain types of ocular surgery, but little has appeared in the literature. This is no doubt due to the marked difficulty in evaluating results and the doubtful efficacy of such therapy. We have employed prednisone and prednisolone preparations locally in 10 cases postoperatively without any untoward effects. The surgeon evaluated these cases as to the amount of postoperative reaction as compared with similar cases in which no corticosteroid was administered. These data are shown in Table 3. None of these patients exhibited marked postoperative iridocyclitis.

Marginal Corneal Ulcers.—Four patients with marginal corneal ulcers were treated with these preparations, and in all the results were excellent. Hogan [7] has shown that corticosteroids play a definite role in the therapy of marginal corneal ulcers. In our series all eyes cleared within five days, and the staining of the cornea had ceased in all cases within two days following institution of local medication. Adjunctive therapy with antibiotic ointment, usually at night only, was included in the therapy of every case, because of the recognized staphylococcal etiology. The results have been tabulated in Table 4.

TABLE 3.—*Postoperative Inflammation*

Case		Diagnosis	Treatment	Response
1	J. T. P.	Following penetrating keratoplasty	Prednisolone alcohol 0.5%	Moderate
2	D. G.	Postoperative partial lamellar keratoplasty	Prednisone, 10 mg. daily	Questionable
3	D. W. T.	Following penetrating keratoplasty	Prednisolone alcohol 0.5% solution q. 2 h.; prednisone 15 mg. daily	Questionable
4	R. McH.	Following partial lamellar keratoplasty with severe eczematoid reaction of eyelid	Prednisone, 40 mg. daily	Reduction of lid swelling in 4 days
5	J. H.	Following partial penetrating keratoplasty	Prednisone acetate 0.1% suspension	Allergic reaction to drug
6	J. J.	Post-trephination	Prednisolone alcohol 0.5%	None apparent
			Subconjunctival injection of 0.25 cc. of prednisone suspension	Slight
7	G. D.	Postcyclodialysis	Prednisolone alcohol 0.5%	Slight
8	K. T.	Post-cataract-extraction	Prednisone acetate 0.5% solution	Moderate
9	A. B.	Post-cataract-extraction	Prednisone 15-30 mg. daily	Slight, if any
10	M. B. B.	Postretinopexy	Prednisone 25 mg. daily	Questionable

TABLE 4.—*Marginal Corneal Ulcers*

Case	Diagnosis	Treatment	Days of Disease	Days to Improvement	Days to Remission	Response
1 E. J.	Secondary to acute catarrhal conjunctivitis	Prednisone acetate 0.5% and oxytetracycline ointment h. s.	2	2	2	Marked
2 J. A. F.	Secondary to acute catarrhal conjunctivitis	Prednisolone alcohol 0.5% and oxytetracycline ointment h. s.	4	1	5	Moderate
3 D. M.	Secondary to acute catarrhal conjunctivitis	Prednisolone alcohol 0.5% and oxytetracycline ointment h. s.	1	1	3	Moderate
4 L. H.	Secondary to old corneal scar	Prednisolone alcohol 0.5% and polymyxin B-bacitracin (Polysporin) ointment	7 (or longer)	1	2	Marked

TABLE 5.—*Miscellaneous Conditions*

Case	Diagnosis	Treatment	Days of Disease	Days to Improvement	Days to Remission	Response
1 J. S. P. Jr.	Pseudomonas keratitis	Prednisolone alcohol 0.5%; Neosporin * ointment, parenteral streptomycin, polymyxin B (Aerosporin), and penicillin	1	3	8	Marked (in conjunction with other medication)
2 J. J.	Seasonal conjunctivitis (both eyes)	Prednisone alcohol 0.1%	1	2	2	Marked
3 J. E. B.	Allergic conjunctivitis (both eyes)	Prednisolone alcohol 0.5%	60	2	4	Marked
4 E. L. V.	Acute follicular conjunctivitis	Prednisolone alcohol 0.5%	1	1	4	Marked
5 M. P. D.	Chronic follicular conjunctivitis	Prednisolone alcohol 0.5%	6 mo.	8	15	Slight
6 L. M.	Chronic follicular conjunctivitis	Prednisolone alcohol 0.5%; prednisone alcohol 0.1%	..	..	..	No change in four days (?)moderate
7 K. L. C.	Vernal conjunctivitis	Prednisone; prednisolone alcohol 0.5%	12 yr.	1	..	None, except slight subjective relief
		Prednisolone alcohol 0.1% suspension	..	1	..	
8 J. F. M.	Reiter's syndrome	Prednisolone acetate 0.5% suspension; prednisone	12 days	..	..	None
9 T. I. G.	Keratitis metaherpetica	Cortisone	4 mo.	..	..	None
		Prednisolone acetate 0.5%	7 mo.	1	15	Marked
10 F. C. K.	Keratitis metaherpetica	Hydrocortisone	3 mo.	..	..	None
		Prednisolone alcohol 0.5%	3 wk.	4	..	Peripheral improvement
		Prednisone acetate 0.5% suspension	..	1	..	Generalized improvement
11 H. H.	Keratitis metaherpetica, both eyes	Hydrocortisone 0.5% suspension	5 mo.	..	..	None
		Prednisolone 0.5%	..	6	6	Marked
12 D. L.	Herpetic keratitis (disciform)	Prednisone; prednisone alcohol 0.1%	7 yr. (recurrent)	2	..	Poor
13 Wm. S.	Central corneal ulcer, recurrent, O. D.	Prednisone alcohol 0.1% ointment	4	..	..	None
		Hydrocortisone 0.5% suspension	..	..	..	None
14 O. B.	Superficial punctate keratitis associated with herpes zoster ophthalmicus	Prednisolone acetate 0.5%; prednisone	6	1	3	Marked
15 N. B.	Acute papillitis	Prednisone	7	4	..	Questionable
16 L. S.	Macular degeneration	Prednisone	4	..	..	Poor
17 B. M.	Macular edema	Prednisone	61	..	..	No change
18 R. B.	Optic neuritis	Cortisone, 120 mg. daily	4 mo.	..	..	None
		Prednisone, 80 mg. with 20 mg. decrease daily to 10 mg.	..	..	..	None
19 M. S.	Retrobulbar neuritis	Prednisone, 20 mg. daily	9	..	..	None
20 W. C. L.	Post-traumatic with anterior chamber hemorrhage	Prednisone, 20 mg. daily; prednisolone alcohol 0.5%	17	1	..	(?) Slight
21 B. J.	Phacotoxic glaucoma	Prednisone, 15 mg. daily; prednisolone alcohol 0.5%	27	1	..	Slight
22 D. K. T.	Chronic congenital glaucoma	Prednisolone alcohol 0.2%	1	1	1	Marked †

* Neosporin is neomycin sulfate, polymyxin B sulfate, and bacitracin.
† See text.

Miscellaneous Conditions.—The remaining patients treated in this series are tabulated in Table 5. As would be expected, allergic conjunctivitis responded extremely well to topical therapy. Chronic follicular conjunctivitis responded poorly or not at all, whereas acute follicular conjunctivitis reacted rapidly, though the follicles remained. Corticosteroid therapy was used with striking results in three cases of keratitis metaherpetica. A patient with disciform keratitis following healed dendritic ulcer suffered a recurrence of the herpetic ulcer at the same time he was receiving topical and oral therapy.

In the case of phacotoxic glaucoma (21, Table 5) the eye whitened rapidly and became less painful, even though the tension remained high. In Case 22 (Table 5), in which a recurrent iridocyclitis with secondary glaucoma was associated with retrolental fibroplasia, topical medication greatly relieved the pain and irritation when applied each hour for the first two days, but when medication was less frequent than this, there was little therapeutic effect. Case 20 (Table 5) responded well to oral and topical medication; however, the tension came under control simultaneously. Oral prednisone was of little value in our cases of retrobulbar neuritis or papillitis. This conforms with the findings of Hogan [7] in his experience with other corticosteroids. One patient with herpes zoster ophthalmicus (Case 13, Table 5) responded rapidly to oral and topical medication; however, the eye was also patched, and a local antibiotic was included in the treatment. With topical prednisolone ointment alone the superficial punctate keratitis continued to progress.

Clinical Laboratory Findings.—In the several cases in which 17-ketosteroid excretion studies were performed, a decrease in the excretion occurred. The eosinophiles dropped markedly in about half the cases receiving oral medication, and in one case no eosinophiles could be found in the specimen examined.

Toxicity.—In this series there were only two patients who developed any untoward reactions that cast suspicion on the corticosteroid medications. The first of these (Case 4, Table 3) was a questionable "moon" facies, but no salt or increased water retention could be demonstrated. One other pation (Case 18, Table 5) revealed a transitory hypochromic anemia and a 1+ guaiac-positive stool; however, no ulcer could be demonstrated by x-rays, and the blood returned to normal spontaneously within 10 days. We were careful, however, to exclude any cases from the series which gave a history of ulcer or diabetes. No hypertension was encountered at the dosage schedule followed, which usually was quite low compared with the dosages used by others in rheumatoid arthritis and other conditions.

Local irritation to the topical administration of these corticosteroid preparations was seldom noted on the two-hour administration schedule. If the time was increased to every hour, the ointment became more irritating. In general, the alcohol form was more irritating, topically, than the acetate form. The suspensions which we employed were prepared in our pharmacy from the sterile crystalline-pure products and required much agitation to form an even suspension. They were momentarily irritating, but none of the patients objected to their use. One patient (Case 5, Table 3) showed allergic reactions to most of the topical preparations, but he had also previously shown allergic responses to all other medications used topically, including corticosteroids.

CONCLUSIONS

Prednisone and prednisolone, used topically in the acetate and free-alcohol forms, exert a pronounced influence on the course of acute inflammation of the anterior segment of the eye. Prednisone, administered systemically, either alone or in combination with the same steroid or prednisolone used locally, produces rapid subsidence of posterior uveitis in many instances. Experimental studies in animals with induced uveitis demonstrated that cortisone was not as effective as prednisone even when cortisone was administered in a strength two and one-

half times that of prednisone. Clinically, several conditions which did not respond favorably to cortisone or hydrocortisone reacted dramatically to prednisone. The indications and contraindications of these new steroids are the same as those previously established for cortisone and hydrocortisone. It is possible, however, that some cases refractory to the earlier corticosteroids will respond to these newer ones. Dramatic improvement occurred in four eyes with severe keratitis metaherpetica. Chronic degenerative conditions do not improve, and these drugs are contraindicated in active tuberculosis, generalized sarcoidosis, diabetes, and dendritic keratitis. Because of the marked subjective response in most cases, careful observation is necessary to avoid the masking of infection and such complications as the occurrence of descemetocele and dendritic ulcer. Systemically, there are fewer side-effects with prednisone than with cortisone and hydrocortisone, although it is therapeutically more powerful. Low maintenance doses are effective.

Although experimental and clinical observations reveal the marked antiphlogistic actions of prednisone and prednisolone in a wide variety of ocular diseases, these drugs deserve further study in other ocular conditions. The effects of subconjunctival and other routes of administration, together with penetration observations, are being studied by other investigators also.

Their rapid action, ability to maintain remissions on low maintenance dosages, and lack of serious toxic effects make these drugs ideally suited to ophthalmic therapeutics. It is possible that these newer synthetic corticosteroids may entirely replace cortisone and hydrocortisone in ophthalmology.

SUMMARY

Prednisone (Meticorten) and prednisolone (Meticortelone), new synthetic steroids, are introduced in the treatment of various inflammatory ocular conditions. Animal experimentation and clinical observations reveal a rapid potent antiphlogistic activity which appears superior, in most instances, to cortisone and hydrocortisone. Several patients who responded poorly, or not at all, to other steroids showed dramatic improvement with prednisone and prednisolone. Toxic effects associated with the systemic use of prednisone are minimal, and small maintenance doses will prevent remissions of many inflammatory ocular diseases. Indications and contraindications are similar to those already determined for cortisone and hydrocortisone. Owing to their rapid potent anti-inflammatory actions and the lack of serious systemic side-effects of prednisone, these new corticosteroids may well supersede previously known steroids in ocular therapeutics.

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Therapy of Congenital Glaucoma

E. NORRIS ROBERTSON Jr., M.D., Oklahoma City

Since Barkan's * reports of the successful treatment of congenital glaucoma by means of goniotomy, which was originally described by de Vincentiis in 1893,[4] many reports confirming this work have been published. Scheie † also has done much original work on congenital glaucoma and has devised another operation, goniopuncture, which, in my hands, has been successful by itself, and in a number of cases in which goniotomy has not.

The technique of these operations has been previously described and will not be given in detail here. In goniotomy the trabecular portion of the angle is incised. In goniopuncture a perforation of the corneoscleral wall is made through the trabecular portion of the angle, and the operation is described by Scheie as a filtering one. There is some controversy concerning the use of a contact glass during these procedures. My own preference is to operate without it.

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From the Department of Ophthalmology, Oklahoma City Clinic and Wesley Hospital.

* References 1 through 3.

† References 5 and 6.

REPORT OF CASES

The first group, of eight cases, includes patients treated by goniotomy (Table 1).

CASE 1.—A white girl, aged 3½ months, was seen in April, 1947. At birth her eyes were noticeably enlarged. Corneal clouding and photophobia appeared shortly after birth. Trephine operations were done on each eye but were not successful in reducing the tension. At the age of 6½ months a goniotomy was done on each eye. The tension normalized almost immediately in the left eye, but, in spite of repeated goniotomies on the right eye, the tension remained elevated. Five years later, the child was seen two days after sustaining a perforating injury to the right eye which required enucleation. The tension in the left eye had remained normal.

CASE 2.—A white boy, seen in March, 1948, at the age of 18 months, had previously had a bilateral trephine operation, without improvement. Goniotomy was done on each eye, also without improvement.

CASE 3.—A white boy was seen a few days after birth, in February, 1950, because of enlarged eyes and cloudy corneas. Goniotomy was done on both eyes within the first two weeks of life. The tension normalized almost at once, and the corneal clouding gradually disappeared within the next few weeks. The tension has remained normal.

CASE 4.—A white girl 17 months old was seen in July, 1951. Her left eye became enlarged and cloudy at the age of 2 months. The right eye was normal. Cyclodialysis, performed elsewhere at the

TABLE 1.—*Results of Goniotomy in Eight Patients*

Case	Age	Sex	Date	Eye	Operation	Result *
1	3½ mo.	F	4/47	O. D.	Goniotomy 4 ×	—
				O. S.	Goniotomy 1 ×	+
2	18 mo.	M	3/48	O. D.	Goniotomy 1 ×	—
				O. S.	Goniotomy 1 ×	—
3	1 wk.	M	2/50	O. D.	Goniotomy 1 ×	+
				O. S.	Goniotomy 1 ×	+
4	17 mo.	F	7/51	O. S.	Goniotomy 1 ×	+
5	5 mo.	M	7/52	O. D.	Goniotomy 1 ×	+
6	3 yr.	M	10/54	O. D.	Goniotomy 1 ×	+
7	1½ mo.	M	8/54	O. D.	Goniotomy 1 ×	+
				O. S.	Goniotomy 1 ×	+
8	5 yr.	M	11/54	O. D.	Goniotomy 1 ×	—

* A plus (+) sign indicates a good result; a minus (—) sign, a poor result.

age of 5 months, did not lower the tension. Goniotomy was done on the left eye, and when the eye was checked one month later, the cornea was clear and the tension normal.

Case 5.—A white boy 5 months old was seen in July, 1952. This case is interesting because the patient was seen so soon after symptoms developed. On July 11, his mother noted that the right eye had become cloudy. Prior to operation, on the following day, the tension was 45 mm. Hg in the right eye and 22 mm. Hg in the left eye. The corneal diameter was 13.5 mm. in each eye. Goniotomy was done on the right eye, with rapid normalization of the tension and clearing of the cornea.

Case 6.—A 3-year-old white boy was seen in October, 1954. The right eye was moderately enlarged, but the cornea was clear. The preoperative tension was 62 mm. Hg in the right eye and 15 mm. Hg in the left eye. Goniotomy was successful in normalizing the pressure.

Cases 7 and 8.—In these cases the glaucoma was associated with naevus flammeus. In Case 7, a 6-week-old white boy was seen in August, 1954, with glaucoma in both eyes. This infant had naevus flammeus involving most of the skin of his face, arms, chest, and abdomen. Involvement of brain tissue placed the clinical picture in the category of the Sturge-Weber syndrome. With the contact glass, a few small blood vessels could be seen on the root of the iris. Goniotomy done on both eyes resulted in immediate normalization of the pressure, with gradual clearing of the corneas over the next four months.

In Case 8, a 5-year-old white boy was seen in November, 1954, with a buphthalmic right eye. The corneal diameter was 14 mm. The child had a small area of naevus flammeus around his involved eye. This was the only area of skin affected, and there was no evidence of involvement of brain tissue. The child's nervous and mental development had been normal. Goniotomy done on the right eye resulted in lowering of the tension for two months, after which it rose to the preoperative level (53 mm. Hg).

The second group, of six cases, includes patients treated by goniotomy and goniopuncture (Table 2).

Case 1.—A 5-year-old white boy was seen in March, 1949. The left eye was markedly buphthalmic and practically blind. The right eye was phthisical, as a result of a perforating injury. Neither goniotomy, done on the left eye at that time, nor goniopuncture, done three years later, was successful in reducing the tension.

Case 2.—A Negro boy aged 4 months was seen in June, 1951. Goniotomy was done on both eyes. One month later, after no noticeable improvement, goniopuncture was done on both eyes, and two weeks later combined goniotomy and goniopuncture were performed on both eyes. After this, the corneas gradually cleared, and the tension has remained normal.

Case 3.—A white boy aged 21 months was seen in July, 1951. The child had marked buphthalmos, congenital cataracts, and a dense leucoma of the right cornea. Goniotomy and goniopuncture, repeated several times on the left eye, were completely unsuccessful. No operation was attempted on the right eye.

Case 4.—A Negro boy aged 4 months was seen in February, 1952. The infant had had cloudy corneas and photophobia for one month. The preoperative tension was 38 mm. Hg in each eye, and the corneal diameters were 13 mm. Goniotomy was

Table 2.—*Results of Goniotomy and Goniopuncture in Six Patients*

Case	Age	Sex	Date	Eye	Operation	Result *
1	5 yr.	M	3/49	O. S.	Goniotomy 1 × Goniopuncture 1 ×	.. —
2	4 mo.	M	6/51	O. D.	Goniotomy 2 × Goniopuncture 2 ×	.. +
				O. S.	Goniotomy 2 × Goniopuncture 2 ×	.. +
3	21 mo.	M	7/51	O. S.	Goniotomy 2 × Goniopuncture 4 ×	— ..
4	4 mo.	M	2/52	O. D.	Goniotomy 1 ×	+
				O. S.	Goniotomy 1 × Goniopuncture 1 ×	+ ..
5	13 mo.	M	8/52	O. D.	Goniotomy 1 × Goniopuncture 2 ×	+ ..
				O. S.	Goniotomy 2 × Goniopuncture 3 ×	+ ..
6	6 mo.	M	3/53	O. D.	Goniotomy 2 × Goniopuncture 2 ×	+ ..
				O. S.	Goniotomy 2 × Goniopuncture 5 ×	— ..

* A plus (+) sign indicates a good result; a minus (—) sign, a poor result.

done on both eyes. Two weeks later goniopuncture was done on the left eye. Following this the pressure was normal in both eyes, but it was several months before all the photophobia disappeared and the corneas became clear.

CASE 5.—A white boy 13 months old was seen in August, 1952. The child had such intense photophobia that he kept his face buried in the bedclothes whenever there was light in his room. His eyes were moderately buphthalmic. The preoperative tension was 41 mm. Hg in each eye, and the corneal diameters were 14 mm. Goniotomy was done on both eyes. The corneas began to clear, but the tension remained above 30 mm. Hg; and goniopuncture was done on both eyes three months later. The tension then was normal in the right eye; but it was necessary to repeat goniotomy, and later do a combined goniotomy and goniopuncture, before the pressure became normal in the left eye. This child has excavation and moderate atrophy of both discs.

CASE 6.—A white boy 6 months old was seen in February, 1953. Glaucoma began at the age of 3 months. His corneas were 14 mm. in diameter, and the tension was 54 mm. Hg in each eye. Goniotomy was done on both eyes. One month later goniopuncture was done on both eyes, followed by combined goniotomy and puncture on both eyes several weeks later. Following all this, the pressure in the right eye was normal, but remained elevated in the left eye. Goniopuncture was done twice more on the left eye before the child was taken to Philadelphia, where goniopuncture was again performed by Dr. H. G. Scheie, but the pressure has remained elevated in the left eye.

The last group includes two cases of patients treated by goniopuncture (Table 3).

CASE 1.—A white girl aged 6 years was seen in November, 1952. Both eyes were markedly buphthalmic and the discs were deeply excavated and atrophic, but she had retained a central visual acuity of 20/50 and a fairly normal peripheral visual field in her right eye and was doing well in the public school. On subsequent observation the intraocular tension varied from the high 20's to the middle 40's. Five months later bilateral goniopuncture was done, with questionable lowering of the tension. Goniopuncture was repeated on each eye eight months later, and miotics were then used. The tension remained in the low and middle 30's for about 10 months. Since that time, with the use of acetazoleamide (Diamox) and miotics, the tension has remained in the high 20's and low 30's. In spite of the poorly controlled tension, there has been no decrease of central vision or loss of peripheral visual field in her right eye during the past two and a half years.

CASE 2.—A 5-year-old white girl was seen in May, 1953, with glaucoma only in her right eye. Goniopuncture normalized the pressure.

COMMENT

Sixteen patients have had the operation of goniotomy and goniopuncture alone or in combination performed on them. A total of 25 eyes were operated on (Table 4). There were eight patients in the first group, treated by goniotomy. One eye of one patient in Group 2 which had only one goniotomy is included in Group 1 for statistical purposes, making a total of 13 eyes. In this group there were nine successes and four failures.

In Group 2 there were six patients for whom goniotomy was not successful and it was necessary to do goniopuncture or the combined operation. There were nine eyes in this group; six were treated successfully and three unsuccessfully.

The two patients in Group 3 were treated only with goniopuncture. Treatment was successful in the eye of a patient with unilateral glaucoma. Treatment was only partially successful in the other patient in that,

TABLE 3.—*Results of Goniopuncture in Two Patients*

Case	Age	Sex	Date	Eye	Operation	Result *
1	6 yr.	F	11/52	O. D.	Goniopuncture 2 ×	(+ —)
				O. S.	Goniopuncture 2 ×	(+ —)
2	5 yr.	F	5/53	O. D.	Goniopuncture 1 ×	+

* A plus (+) sign indicates a good result; (+ —) indicates that tension was poorly controlled but visual loss was stopped.

TABLE 4.—*Summary of Results*

	No. of Eyes	Tension Remaining Elevated	Tension Normalized
Group 1 (goniotomy)	13	4	9
Group 2 (goniotomy and goniopuncture)	9	3	6
Group 3 (goniopuncture)	3	2	1
Totals	25	9	16

while the visual loss was arrested, the tension was not well controlled.

The results of treatment were, of course, much better when the disease was detected early. Ten of the eyes in this series showed moderate to marked buphthalmos and optic atrophy, and only 3 of these eyes were cured. However, treatment was completely successful in 13 of the 15 eyes which were nonbuphthalmic.

Much has been written concerning the presence of mesoblastic tissue blocking the trabecular portion of the angle in congenital glaucoma. In my experience it has been difficult to detect, and I agree with Scheie ‡ in his statement that he noted little, if any, difference in the appearance of the angles of eyes with congenital glaucoma and those of normal children of the same age. If the presence of mesoblastic tissue in the angle is ruled out, then the operation of goniotomy does not remove a mechanical obstruction, and the good results following its use will have to be explained in some other way.

As stated before, there are many reports on successful goniotomies, but, except for Scheie's § reports, and an earlier one of mine,[7] not a great deal has been written on successful goniopunctures. In my opinion goniotomy is the operation of choice in most cases of congenital glaucoma, particularly the early ones, but there are a significant number of cases in which goniotomy alone is not successful and in which goniopuncture or the combined operation is then required. Both these operations are safe, are reasonably easy to perform, can be repeated several times, and do not change the appearance of the eye.

‡ Scheie, H. G.: Personal communication to the author.

§ References 6 and 8.

SUMMARY

The cases of 16 patients with congenital glaucoma treated by goniotomy, goniopuncture, or a combined goniotomy and goniopuncture are reported. Twenty-five eyes were treated. Treatment was successful in 16 eyes, unsuccessful in 7 eyes, and partially successful in 2 eyes.

391 N. W. 12th St.

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Report on Thirteen Eyes Treated by Goniopuncture

GEORGE S. TYNER, M.D.
and
EDWARD J. SWETS, M.D., Denver

Thirteen eyes (nine patients), 8 with congenital glaucoma, 2 with juvenile glaucoma, 2 with congenital aniridia and glaucoma, and 1 with glaucoma secondary to surgery of congenital cataract, were treated by goniopuncture. This procedure was chosen because of its simplicity and infrequency of operative and postoperative complications, and because the advanced stage of many of the eyes almost precluded the successful employment of other procedures.

The procedure as described by Scheie,[1] in 1950, was employed in all cases. No operative complications were encountered. The procedure was successful in controlling the tension in nine eyes. It failed in two eyes and was of questionable value in two eyes with congenital glaucoma. The success or failure of the procedure in the two eyes questioned could not be definitely determined because at the date of follow-up the infant was too ill to withstand general anesthesia. Among the nine eyes successfully treated can be included the two eyes with juvenile glaucoma, the two with aniridia, the one eye with secondary glaucoma, and four of the eyes with congenital glaucoma.

The criterion for a cure or control was arbitrarily taken as a tension below 30 mm. of mercury in the absence of miotics. Tensions were determined with a Schiøtz tonometer under general anesthesia wherever the age of the patient made it necessary. The Table summarizes the findings in all cases.

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From the Glaucoma Clinic of the Division of Ophthalmology, Department of Surgery, University of Colorado School of Medicine.

REPORT OF CASES

CASE I.—H. F. This 25-year-old white woman was first seen on March 17, 1954, through the courtesy of Dr. Ralph Danielson and Dr. J. C. Long, of Denver. She gave a history of bilateral congenital glaucoma, first diagnosed at the age of 3 months. Her right eye had been eviscerated in

Results of Goniopuncture

Case	Age	Eye	Stage	No. Oper.	Follow-Up Time	Final Tension, Mm. Hg	Results*	Type
1 H. F.	25 yr.	O. S.	Advanced	1	12 mo.	28	+	Congenital
2 J. R.	12 yr.	O. D.	Early	2	13 mo.	20	+	Juvenile
		O. S.	Early	2	14 mo.	28	+	Juvenile
3 D. M.	13 mo.	O. D.	Advanced	2	8 mo.	28	+	Congenital
		O. S.	Advanced	2	8 mo.	38	—	Congenital
4 D. S.	5 yr.	O. S.	Advanced	3	9 mo.	29	+	Congenital
5 L. K.	6 yr.	O. S.	Advanced	1	14 mo.	17	+	Congenital
6 L. G.	9 mo.	O. D.	Advanced	1	5 mo.	?	—?	Congenital
		O. S.	Advanced	1	4 mo.	?	—?	Congenital
7 G. A.	4 mo.	O. D.	Early	2	6 wk.	54	—	Congenital
8 P. M.	7 yr.	O. D.	Early	1	7 mo.	20	+	Aniridia
		O. S.	Early	1	7 mo.	24	+	Aniridia
9 S. O.	18 mo.	O. D.	Advanced	2	1 mo.	28	+	Secondary

* A successful result is indicated by +; a failure by —.

1933 because it was buphthalmic, blind, and painful. The remaining, left eye had undergone two cyclodialyses and an iridencleisis.

Ophthalmologic Examination.—Vision: Vision was 20/200 with and without glasses.

External Examination: The eye was photophobic, lacrimating, and irritable. There was a deep elevated central corneal leucoma. The lens was opaque at the site of the iridencleisis superiorly. The horizontal corneal diameter was approximately 14 mm.

Ophthalmoscopic Examination: Owing to the corneal haze, the fundus could not be seen.

Gonioscopic Examination: Although the anterior chamber was deep, there was an almost complete disorganization of the angle, with closure, due to peripheral anterior synechiae, except for one area at 2 o'clock and one area at 5 o'clock.

Visual Field Examination: Unreliable, due to photophobia and blepharospasm.

Ocular Tension: Ocular tension was 41 mm. Hg (Schiøtz).

Clinical Course.—Two goniopunctures were done at the same operation on March 17, at the open sites of the angle at 2 and 5 o'clock. The operative and postoperative courses were uneventful. The eye was somewhat irritable for one month postoperatively. On July 20, the eye was white and quiet and the patient comfortable. On Jan. 25, 1955, the eye again became irritable, but the irritation subsided on use of 2.5% hydrocortisone locally. When last examined, on March 28, 1955, the eye was white and quiet and the ocular tension was 28 mm. Hg (Schiøtz). The visual acuity was 20/200. This eye is included to illustrate that the procedure can yield a successful result in a severely damaged and previously surgically treated eye.

Case 2.—J. R., a 12-year-old white boy, was first seen on April 15, 1953. He was brought to the Clinic by his family because his father, paternal uncle, paternal grandfather, and cousin had had glaucoma at an early age. Other members of the family had poor vision, but a definite diagnosis of glaucoma had not been established. He had no ocular complaints other than some irritability.

Ophthalmologic Examination.—Vision: The vision was 20/20 in each eye.

External Examination: External examination revealed no abnormality. Both corneas were clear, and the anterior chambers were of normal depth. No corneal enlargement was noted.

Ophthalmoscopic Examination: The media were clear. The optic discs showed moderate cupping, but only slightly more than physiological.

Gonioscopic Examination: The angle of each eye was open. The trabecular regions were characterized by the presence of heavy pigment and some abnormal iris processes similar to pectinate ligament remnants.

Visual Field Examination: Visual fields were normal in both eyes.

Ocular Tension: The ocular tension was 55 mm. Hg in each eye.

Clinical Course.—A trial of various miotics in each eye was unsuccessful in controlling the tension. Bilateral goniopunctures were done on July 8, 1953, at Children's Hospital, in Philadelphia, without operative or postoperative complication. He was reexamined under general anesthesia four months later (Nov. 12). The tension of the right eye was 30 mm. Hg and that of the left eye 46 mm. Hg (Schiøtz). A second goniopuncture was done on the left eye. The previous puncture appeared open by gonioscopy. He was next examined on Dec. 31, 1953. The tension of the right eye was 45 mm. Hg and that of the left eye 33 mm. Hg (Schiøtz). A second goniopuncture was done on the right eye. The first puncture appeared open by gonioscopy. Both goniopunctures appeared open in the left eye. The patient was last examined on Jan. 20, 1955, under general anesthesia. The tension of the right eye was 20 mm. Hg and that of the left eye 28 mm. Hg (Schiøtz). The vision and visual fields have remained normal. Both eyes are included among the successful results. This case illustrates that the tension was uncontrolled despite apparently patent punctures. Repetition of the procedure was required to obtain a successful result.

Case 3.—D. M., a 13-month-old white female infant, was first seen on Feb. 2, 1954. She was referred by Dr. Richard C. Vanderhoof, of Colorado Springs, Colo., because of bilateral congenital glaucoma. Bilateral cyclodialysis and iridectomy had failed to control the tension in either eye.

Ophthalmologic Examination.—Vision: The child responded to light and hand movements with each eye.

External Examination: Both corneas were hazy and the patient extremely photophobic. The corneal diameter was 14.5 mm. in the horizontal meridian in each eye. The pupils were relatively small, due to the frequent use of miotics.

Gonioscopic Examination: The angle of each eye was open and the trabecular space free of abnormal tissue. The pigment at the root of the iris appeared scalloped and the entire iris somewhat atrophic, with dilated vessels. A peripheral anterior synechia was present at the site of the cyclodialysis in each eye. Bilateral peripheral iridectomies were open. Some of the corneal haze in both eyes could be accounted for by ruptures in Descemet's membrane.

Ophthalmoscopic Examination: Ophthalmoscopic examination showed marked glaucomatous cupping in both eyes.

Ocular Tension: The initial tension with the patient under general anesthesia following the fre-

quent use of miotics was 25 mm. Hg in the right eye and 38 mm. Hg in the left eye (Schiøtz).

Clinical Course.—A goniopuncture was done on the left eye on Feb. 24, 1954. The child was reexamined on March 2, 1954, without miotics. The tension of the right eye was 38 mm. Hg and that of the left eye 33 mm. Hg (Schiøtz). A goniopuncture was then done on the right eye. The child was next examined under general anesthesia four and a half months later (July 19). The tension of both eyes was 35 mm. Hg (Schiøtz). Both previous punctures were closed or partially closed by peripheral anterior synechiae. Bilateral goniopunctures were repeated on this date. The child was next seen seven months later (Feb. 10, 1955). The tension of the right eye was 28 mm. Hg and that of the left eye 38 mm. Hg (Schiøtz). A third goniopuncture was done on the left eye. The right eye is included among the successful results. The left eye is considered a failure, since no further follow-up has been obtained. It illustrates that failure of the procedure could be explained on the basis of peripheral anterior synechiae.

CASE 4.—D. S., a 4½-year-old white boy, was first seen on Dec. 3, 1953, with a history of congenital glaucoma of the left eye since birth. The right eye had always been normal. He was seen through the courtesy of Dr. James P. Rigg, of Grand Junction, Colo. He had not previously been operated on. The tension of the left eye had been 70 mm. Hg without miotics and 40 mm. Hg with miotics.

Ophthalmologic Examination.—Vision: The vision of the right eye was 20/30 and vision of the left eye counting fingers.

External Examination: The right eye was normal throughout. The left cornea was hazy, the eye buphthalmic, and the corneal diameter 14 mm.

Ophthalmoscopic Examination: Both fundi appeared normal.

Gonioscopic Examination: The angle of the left eye was open. The trabecular region showed iris-like tissue extending up from the root of the iris to the area of Schwalbe's line. It is interesting to note that the normal, right eye had similar tissue but did not have glaucoma.

Ocular Tension: The tension of the right eye was 18 mm. Hg and that of the left eye 40 mm. Hg (Schiøtz).

Clinical Course.—On Jan. 4, 1954, a goniopuncture was done on the left eye. There were no operative or postoperative complications. A filtering subconjunctival bleb was formed at the site of the puncture. The child was subsequently examined under general anesthesia on Feb. 24, 1954. Despite the fact that the previous puncture appeared open by gonioscopic examination, the tension was 38 mm. Hg. No filtering bleb could be observed. A second goniopuncture was performed. The child was reexamined five months later (July 29), and, again, both punctures appeared open by gonioscopic examination but the tension was 40 mm. Hg. A third goniopuncture was performed. Subsequent tension readings are recorded as follows: 25 mm. Hg on Aug. 25, 1954; 15 mm. Hg on Nov. 1, 1954; 17 mm. Hg on Jan. 4, 1955, and 20 mm. Hg on April 11, 1955. There was a diffuse subconjunctival bleb inferiorly at the site of the punctures nine months after the last procedure. The visual acuity in the eye operated on improved to 20/50. The left optic disc appeared slightly cupped temporally and paler than the right. This case is included among the successful results and illustrates that three punctures were required to normalize the tension. No cause for failure of the first two procedures could be recognized. Both punctures appeared patent by gonioscopy.

CASE 5.—L. K., a 6-year-old white boy, was first seen on Feb. 11, 1954, through the courtesy of Dr. John C. Long, Denver, and Dr. George Brown, of Fort Collins, Colo. A diagnosis of bilateral congenital glaucoma had been made at the age of 7 months, in July, 1948, when the ocular tension was 65 mm. Hg (Schiøtz). The child received a perforating ocular injury in the right eye in December, 1951.

Ophthalmologic Examination.—Vision: There was no light perception in the right eye. The vision of the left eye could not be accurately determined but was estimated at 20/400 to 20/200.

External Examination: The right eye was blind and phthisical. It was shrunken in size, as a result of the perforating injury. The left eye was moderately photophobic and the cornea moderately hazy. The corneal diameter was 14 mm.

Ophthalmoscopic Examination: The fundus of the right eye could not be seen. The left eye had clear media except for some corneal haze. The disc was deeply cupped and atrophic.

Gonioscopic Examination: The angle of the left eye was open. No abnormality could be seen in the trabecular region. The cyclodialysis cleft was closed.

Ocular Tension: The tension of the left eye was 30 mm. Hg (Schiøtz), with the patient under general anesthesia. The pupil was small, due to the frequent instillation of miotics for an indefinite period prior to the examination.

Clinical Course.—On Feb. 12, 1954, the right eye was enucleated and a goniopuncture done on the left eye. There were no operative complications. The child was placed on instillations of 2.5% of hydrocortisone ophthalmic suspension five times daily and discharged from the hospital on the fourth postoperative day. On the seventh postoperative day the child returned to the hospital because of an acutely painful left eye. The hydro-

cortisone had not been used at home. The tension was 50 mm. Hg (Schiøtz). It was believed that this rise in tension represented a secondary glaucoma due to plasmoid aqueous. The patient was treated with local instillations of hydrocortisone and pilocarpine. The tension was reduced to 29 mm. Hg (Schiøtz) within 24 hours. Subsequent tension determinations with the child still on pilocarpine and hydrocortisone were 28 mm. Hg on March 5, 1954, and 26 mm. Hg on April 17, 1954. Follow-up examinations on July 29, 1954, and April 11, 1955, without miotics or hydrocortisone, showed the tension to be 17 mm. Hg (Schiøtz). There was a grossly visible filtering bleb at the site of the puncture. This case represents a 14 months' cure with one goniopuncture on an eye with advanced glaucoma unsuccessfully treated by cyclodialysis.

CASE 6.—L. G., a 9-month-old infant, was first seen on Feb. 28, 1954, through the courtesy of Dr. James P. Rigg, of Grand Junction, Colo. He had a history of bilateral congenital glaucoma. Bilateral trephinations had been done, without controlling the tension.

Ophthalmologic Examination.—External Examination: Despite the bilateral trephinations and frequent use of miotics, the child was extremely photophobic, the corneas hazy, and the corneal diameters enlarged to 14 mm. in each eye. Bilateral trephination blebs were visible at 12 o'clock.

Vision: The vision acuity could not be obtained.

Ophthalmoscopic Examination: No view of the fundus could be obtained, owing to the presence of the small pupils and a cataract in the right eye.

Gonioscopic Examination: The angle of each eye was open. No abnormal trabecular tissue could be recognized.

Ocular Tension: Miotics were discontinued for one month. The ocular tension of the right eye was 40 mm. Hg and of the left eye 35 mm. Hg (Schiøtz) with the patient under general anesthesia.

Clinical Course.—Goniopunctures were performed on Feb. 23, 1954 (left eye), and March 25, 1954 (right eye). Following goniopuncture, during the postoperative period, the corneas were clear but after several days again became cloudy. The patient was then placed on treatment with pilocarpine and topical cortisone drops. The child was readmitted to the hospital for follow-up examination four months later, July 28. At this time he was still on miotics and local cortisone. Both corneas were clear, and the child was not photophobic. This was in contrast to his appearance before the goniopunctures were done. Prior to goniopuncture, the corneas had been hazy and the eyes extremely photophobic, despite miotics and bilateral trephination. Pediatric examination disclosed a low-grade fever and a congenital kidney abnormality, diagnosed as tubular nephropathy. It was the opinion of the pediatrics department that the prognosis for the life of the child was extremely limited and that he should not be subjected to general anesthesia. This case is included because it illustrates that at least a clinical improvement was noted following the use of goniopuncture. Miotics and bilateral trephination had not been successful in controlling the tension prior to this procedure.

CASE 7.—G. A., a 3-month-old white boy, was first seen on Dec. 14, 1954, through the courtesy of Dr. William Droegemueller, of Greeley, Colo. The right cornea had been slightly hazy since birth, and the left cornea had been normal. General examination showed a normal-appearing, healthy infant with a midline naevus flammeus over the occiput and forehead. Similar lesions appeared on the neck and trunk. Neurological examination was negative, and roentgenological examinations were negative for evidence of cerebral angiomas or calcifications.

Ophthalmologic Examination.—Vision: The patient followed a light with each eye.

External Examination: The right cornea was hazy and measured 13.5 mm. in the horizontal meridian. The left eye appeared normal. The corneal diameter was 11 mm.

Ophthalmoscopic Examination: The fundus could not be observed in the affected eye because of corneal haze. The fundus in the normal eye showed no evidence of pathology.

Gonioscopic Examination: Owing to the corneal haze, the angle detail could not be observed in the affected eye. The angle of the unaffected eye was normal.

Ocular Tension: With the child under general anesthesia, the tension of the right eye was 40 mm. Hg (Schiøtz), and the tension of the left eye was 25 mm. Hg (Schiøtz).

Clinical Course.—On Dec. 17, a goniopuncture was done, without incident, on the right eye. The cornea was cloudy on the first postoperative day and remained cloudy during the entire postoperative observation period. The child was again examined under general anesthesia on Feb. 4, 1955. The tension of the right eye was 37 mm. Hg (Schiøtz). A goniopuncture was repeated, without operative complication. The cornea cleared immediately after the puncture, and the iris could be visualized. The stroma appeared atrophic and marked by the presence of numerous enlarged vessels. The postoperative course was as it had been after the first operation. Topical hydrocortisone and atropine used after the first operation were ineffective, as were topical cortisone and pilocarpine following the second operation, in preventing postoperative reaction. The child was reexamined under general anesthesia on March 17, 1955. The tension was 54 mm. Hg (Schiøtz). A goniotomy was done over the inferior nasal quadrant. The

procedure was discontinued when the anterior chamber emptied. The first postoperative day was marked by the same reaction of hazy cornea and apparently increased tension. The child has since been placed on 10 mg. of acetazoleamide (Diamox) per kilogram of body weight, pilocarpine t. i. d., and physostigmine at bedtime. At the last examination, on April 11, 1955, the tension is still uncontrolled. This case represents a failure for goniopuncture, goniotomy, acetazoleamide, and miotics.

CASE 8.—P. M., a 6½-year-old white girl, was first seen on June 25, 1954, through the courtesy of Dr. Walter Dardis, of Pueblo, Colo. She had a history of bilateral congenital aniridia with secondary glaucoma and lens opacities.

Ophthalmologic Examination.—External Examination: Both corneas were slightly hazy but not enlarged. No iris tissue was grossly visible in either eye.

Vision: The estimated visual acuity was 20/400 to 20/200 in each eye.

Ophthalmoscopic Examination: The fundi were poorly seen, owing to corneal haze and lens opacities. The discs appeared slightly paler than normal, with some cupping.

Gonioscopic Examination: The anterior chamber angle was open in each eye. Rudimentary fragments of iris could be seen in all meridians in both eyes. The ciliary body and suspensory ligament of the lens were clearly visible.

Ocular Tension: The tension of the right eye was 55 mm. Hg, and the tension of the left eye was 52 mm. Hg (Schiøtz).

Clinical Course.—For investigational purposes, the child was placed on 10 mg. of acetazoleamide per kilogram of body weight. The tension was reduced to 37 mm. Hg in the right eye and 40 mm. Hg in the left eye (Schiøtz). No significant further reduction was noted after the addition of miotics. On July 29, 1954, during the teaching session of the Summer Conference of the Colorado Ophthalmological Society, bilateral goniopunctures were done by Dr. Harold G. Scheie. The operative and postoperative courses were uneventful. Follow-up examintion six and a half months later (Feb. 10, 1955) showed a tension of 20 mm. Hg, right eye, and 24 mm. Hg, left eye (Schiøtz). The child had received no miotics or acetazoleamide during the interim. This case represents a bilateral aniridia successfully treated with one goniopuncture on each eye.

CASE 9.—S. O., a white female infant of 18 months, was first seen on Dec. 17, 1954, through the courtesy of Dr. Ralph Danielson, of Denver. The child had a history of bilateral congenital cataract, with normal fundi and anterior chamber angles. The right eye had been operated on several occasions for congenital cataract. For some obscure reason, even though the lens capsule and cortex were deeply incised, there was no absorption of the lens material. Instead of the usual swelling and fluffy appearance of the lens substance following needling, the lens remained intact, showing only the deep lines of incision. The appearance of the lens material after incision could be compared to suet. Because of the failure of absorption, it was necessary to remove the lens material by linear extraction. The dense capsular membrane required subsequent iridocapsulotomy. As a result of these procedures, secondary glaucoma had developed from peripheral anterior synechiae. A cyclodialysis had failed to control the tension.

Ophthalmologic Examination.—Vision: The patient followed a light with each eye.

External Examination: The right cornea was hazy. The horizontal meridian measured 13 mm. The eye was aphakic, with some fragments of lens capsule remaining. The left eye was normal except for the congenital cataract. The corneal diameter was 12 mm.

Ophthalmoscopic Examination: Both fundi appeared normal.

Ocular Tension: The tension of the right eye was 50 mm. Hg, and the tension of the left eye was 20 mm. Hg (Schiøtz).

Gonioscopic Examination: The angle of the anterior chamber of the right eye was almost entirely closed by peripheral anterior synechiae except for a small area at 7 o'clock and another at 9 o'clock. On Dec. 17, 1954, a goniopuncture was done at 7 o'clock through the open portion of the angle. Subsequent examination, with the child under general anesthesia, on Jan. 12, 1955, showed the puncture to be above Schwalbe's line and nonfunctioning. The tension was 54 mm. Hg (Schiøtz). A goniopuncture was done in the other, free portion of the angle. On the second postoperative day, at the time of dressing, it was noted that the protective shield had been displaced and the eye traumatized. A massive hyphema was present. Subsequent examination, on Feb. 16, with the child under general anesthesia, showed a deposition of blood pigment on the endothelial surface of the cornea, but the ocular tension was 28 mm. Hg (Schiøtz).

Although the follow-up period is too short to determine if this procedure will be permanently effective in controlling the tension, this case is included to illustrate that a goniopuncture can be done on an eye with the angle almost completely occluded by peripheral anterior synechiae without undue operative risk. It is also of interest that the goniopuncture remained functioning despite the presence of blood in the anterior chamber for a period of several weeks. The cyclodialysis, which had been done prior to the goniopuncture, had gone without undue operative difficulty but had failed.

COMMENT

Goniopuncture has been used for the treatment of 13 eyes with glaucoma. Eight had congenital glaucoma; two had juvenile glaucoma; two had congenital aniridia with glaucoma, and one had glaucoma due to peripheral anterior synechiae following repeated surgery for the treatment of congenital cataract. All eyes were characterized by deep chambers and open anterior chamber angles except in the infant with secondary glaucoma and a 25-year-old patient with advanced congenital glaucoma. The operation failed in two eyes with congenital glaucoma and was of questionable value in two eyes with congenital glaucoma. Of the latter two eyes, no statement of proof can be made as to the control of the tension, but the patient showed a clinical improvement, as indicated by clear corneas and lack of photophobia and blepharospasm. The tactile tension in each eye, although unreliable, seemed normal. The operation was successful in the nine remaining eyes, eight of which have been followed for a period of from 7 to 14 months. For the present follow-up period, this represents a 64% success in controlling the ocular tension. The two eyes with clinical improvement but no follow-up tensions are not included as successful.

Of interest is the number of procedures necessary on each eye to control the tension. Each eye with congenital aniridia required only one goniopuncture to control the tension. The two eyes with juvenile glaucoma required two punctures each to control the tension. The cause for failure of the first puncture was not apparent. Advanced congenital glaucoma in a 25-year-old woman, who had been subjected to numerous other operative procedures, was controlled with two goniopunctures done at the same operation. Two punctures were attempted at this time because there were only two small areas in the anterior chamber angle which were free of peripheral anterior synechiae. The infant with secondary glaucoma following needling of a congenital cataract likewise received two punctures, but at different operations. These two punctures were made in the only two areas which were not closed by peripheral anterior synechiae. It could be observed that the first puncture failed because the operative procedure was faulty. The puncture was made at a point above Schwalbe's line rather than in the trabecular space. It is interesting to note that the second procedure was successful despite the fact that the patient sustained a traumatic hyphema two days after surgery, due to a blow on the eye.

A 13-month-old infant with bilateral congenital glaucoma had been subjected to cyclodialysis and irridectomy on both eyes prior to this survey. One eye, which was successfully controlled, was operated on twice by goniopuncture, and one eye, which was not controlled, was operated on twice. The result of the third puncture on the latter eye has not been finally evaluated. The cause for failure of the first puncture on each eye is closure by peripheral anterior synechiae. One 5-year-old child with advanced congenital glaucoma in one eye had not had any previous surgery. This eye required three goniopunctures before the tension was controlled. The cause for failure of the two first punctures could not be determined. Both punctures appeared open by gonioscopic examination, prior to the last procedure.

These results are in accord with Scheie's original observation [1] that the stage of glaucoma seems of little importance in influencing the outcome of this operation. Eight eyes operated on were in the advanced stage before the operation was attempted. Five were successfully treated; one was a failure, and the results in two were of doubtful value. Five of the operations were in the early stage. Four were successful, and one failed. The failure occurred in one eye which was not subsequently controlled by goniotomy, acetazoleamide, and miotics.

From this short experience, it seems apparent that one of the principal merits of this procedure is that it can be employed in advanced cases with little risk of serious hemorrhage, damage to the lens, or severe postoperative reaction. A further advantage in this procedure is that its repeated employ-

ment does not preclude the subsequent use of a goniotomy or a filtering operation should the repeated attempts with a goniopuncture fail to control the tension.

Topical hydrocortisone postoperatively may aid in reduction of tension in cases with a secondary rise in tension due to plasmoid aqueous. Pilocarpine instead of atropine may be indicated postoperatively to prevent the formation of peripheral anterior synechiae.

SUMMARY AND CONCLUSION

Goniopuncture was effective in controlling the ocular tension in 9 of 13 eyes.

The stage of the disease was not a significant factor in the success of the operation in this series.

The operation was successful in patients ranging from 1 to 25 years of age.

Repeated operations were necessary in some eyes to control the tension despite the apparent patency of previous punctures. The cause for failure of apparently open punctures remains obscure.

Peripheral anterior synechiae following two punctures and faulty placement of a puncture above Schwalbe's line in one case were recognized as causes for failure.

No operative complications were encountered in 22 goniopunctures. No serious postoperative complications were recognized. Temporary rises in tension postoperatively were not unusual.

We do not feel that final evaluation can be made in any of our cases until further follow-up can be obtained.

REFERENCE

1. Scheie, H. G.: Goniopuncture: A New Filtering Operation for Glaucoma, A. M. A. Arch. Ophth. **44**:761 (Dec.) 1950.

Mechanical Changes During Accommodation Observed by Gonioscopy

HERMANN M. BURIAN, M.D.
and
LEE ALLEN, Iowa City

It is relatively easy to observe the mechanical changes taking place during accommodation in the centrally located parts of the structures of the anterior segment of the eye. These changes have been carefully studied and form the basis of the various theories of accommodation. But such investigations could indicate only indirectly what, if any, specific changes were occurring in the peripheral parts of the structures involved, since direct observation was difficult or impossible with ordinary means of examination. Direct observations were possible only in cases of gross anatomic, and probably also functional, abnormalities, such as colobomas of the iris or aniridia. Even in these circumstances the chamber angle was not accessible to direct study. It is, however, of interest and importance to have a detailed knowledge of the behavior during accommodation of the periphery of the lens and iris and of the trabecular band, all of which are intimately concerned in the activities of the zonule of Zinn and the ciliary body.

The advent of gonioscopic contact glasses and prisms has made these regions visually much more accessible than heretofore; and for the study of mechanical changes occurring in them the gonioscopic devices which permit slit-lamp illumination are most helpful, since contours of planes of discontinuity between otherwise transparent tissues can be studied well only by such means. Another advantage of gonioscopy lies in the exaggeration of contour changes of some of the structures, due to foreshortening.

For all these reasons, we have undertaken a gonioscopic investigation of normal eyes during active accommodation and during relaxation. This paper is a first report on our findings.

METHOD OF EXAMINATION AND CHOICE OF SUBJECTS

All subjects were observed on a Haag-Streit slit lamp and by means of an Allen-Thorpe gonioprism,[1] which has relatively greater maneuverability than similar devices. Since the eye wearing the gonioprism could not be used for fixation, it was necessary for the subject to use his unhampered eye for this purpose. It was assumed that mechanical changes occurring during relaxation and active accommondation would be reflected in the fellow eye under gonioscopic observation. It was furthermore necessary to arrange an appropriate angle of separation between the distant and the near fixation objects to balance as nearly as possible the reflex convergence and divergence of the eye during changes of fixation.

A special attachment was built which could be mounted upon the housing of the biomicroscope of the slit lamp. It consisted essentially of a mirror placed before the fixating eye, a near fixation object, and a slot next to it through which the patient could look at a distant object (Fig. 1). This slot was movable in the plane of the near fixation object and could be so adjusted that the convergence movement attendant upon accommodation was minimized. The visual distance of the near object was 20 cm. A clip before the mirror allowed the intro-

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From the Department of Ophthalmology, State University of Iowa College of Medicine.

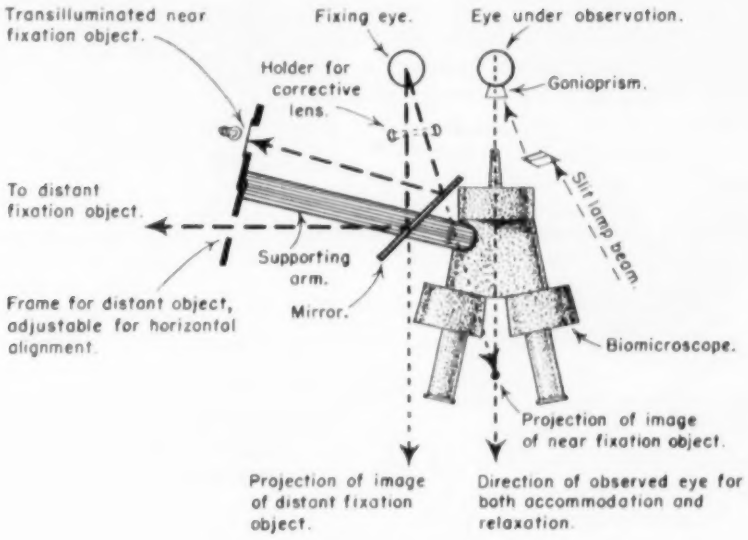

Transilluminated near fixation object.
Fixing eye.
Eye under observation.
Holder for corrective lens.
Gonioprism.
To distant fixation object.
Slit lamp beam.
Supporting arm.
Frame for distant object, adjustable for horizontal alignment.
Mirror.
Biomicroscope.
Projection of image of near fixation object.
Projection of image of distant fixation object.
Direction of observed eye for both accommodation and relaxation.

Fig. 1.—Diagram of fixation device.

duction of spherical lenses when it was necessary to correct a refractive error of the subject.

Twenty subjects between the ages of 15 and 26 years were selected at random from routine refraction patients. This age group was considered to be most suitable for our purpose because the development of the eye should be complete, yet accommodation still quite active. Subsequently, a comparative study will be conducted in older subjects.

RESULTS

1. *Pupil and Pupillary Margin of Iris.*—A constriction of the pupil was noted in all cases. This neither affirms nor denies that the pupillary contraction in the near-vision complex is coupled with accommodation, since no attempt was made to exclude rigorously all convergence movements.

The forward movement of the pupillary margin and the adjacent iris was also noted, although it could not be seen directly. It was inferred from the following observation: A simple centripetal movement of the pupillary margin along the surface of a stationary lens would result in a noticeable displacement of the margin toward the edge of the gonioscopic mirror, owing to the geometric projection of the gonioscopic image. Actually, however, when the pupil constricted during accommodation, it remained at the same level in the gonioscopic view. This could occur only if the pupillary margin had moved forward as well as toward the anatomic axis of the eye (Fig. 2).

2. *Iris.*—During accommodation the iris rolls flattened out on top, possibly owing to stretching of the iris tissue by constriction of the sphincter muscle, but the furrows seemed, nevertheless, to retain their sharpness in the majority of cases.

The most remarkable change was seen in the middle one-third of the body of the iris. This part of the iris bowed backward during active accommodation, forming a deep hollow (Fig. 3*A*), and returned to its normal position when the eye was relaxed (Fig. 3*B*). In many young people this bowing back was so pronounced that it could readily be observed in the upper and lower parts of the iris with the slit-lamp beam even without the use of the gonioprism. The portion of the iris peripheral to the part just described, and all the way to the root, could be seen to move slightly backward, thus opening the angle slightly.

Helmholtz [2] had already noted the backward displacement of the iris periphery in accommodation. He considered that it was

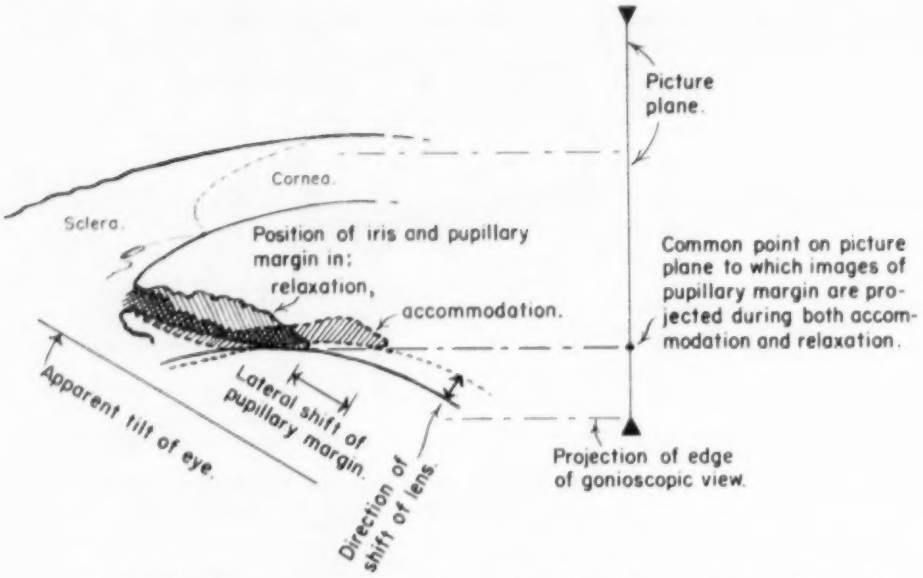

Picture plane.
Cornea.
Sclera.
Position of iris and pupillary margin in:
relaxation,
accommodation.
Common point on picture plane to which images of pupillary margin are projected during both accommodation and relaxation.
Apparent tilt of eye.
Lateral shift of pupillary margin.
Direction of shift of lens.
Projection of edge of gonioscopic view.

Fig. 2.—Schematic cross section of chamber-angle region of the eye showing shifting of iris and pupillary margin, and direction of movement of anterior surface of lens (without intention of showing extent) during changes in accommodation.

necessitated by the displacement of the aqueous from the central part of the anterior chamber, owing to the narrowing of the chamber in this region by the bulging of the lens. We think that the sinking back of the iris is made possible by reduction in the tension of the zonular fibers all the way to their anterior insertions in the ciliary body, and to the reduction in the horizontal diameter of the lens during accommodation. Thus, the iris can be pushed backward in its peripheral part by the aqueous pressed away from the center of the anterior chamber.

3. *Ciliary Body Band.*—In most subjects there were some changes in the ciliary body band, but these were not consistent. The delicate texture of the fibers of the iris root and of the inner uveal trabecular fibers could be seen to change in most patients, as if these tissues were being shifted slightly in position. However, the change was subtle and difficult to describe. Attempts to determine whether the ciliary body band broadened or narrowed (independently of the width of the anterior chamber as a whole) were not successful.

In a 24-year-old woman the contour of the slit beam over the ciliary body differed in relaxation and in accommodation. During relaxation the line of the slit beam across the ciliary body had much the same curve as the line across the trabecular band and the posterior corneal surface (Fig. 4*A*). During accommodation the slit-beam line became more curved, especially nearest the root of the iris. This change might have resulted from tension put by the iris root on the inner uveal fibers, which were pulled across the apex of the chamber angle. Or it might have been caused by a thickening of the meridional ciliary muscle fibers at that level, or by a combination of the two factors.

4. *Scleral Spur.*—Because of an initial movement of the image at the instant of change of fixation, and before vergence for the distance was established, it was impossible to decide whether there was any change in the position of the scleral spur. Its width, color, and texture seemed to remain constant.

5. *Trabecular Zone and Ring of Schwalbe.*—The available evidence indicates that there

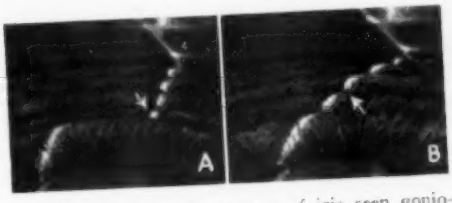


Fig. 3.—Changes in contour of iris seen gonioscopically during changes in accommodation.

A, during active accommodation. Arrow points to midportion of iris, which is being pressed backward.

B, during relaxation of accommodation. Arrow points to slit-beam section of iris surface, bowed very slightly forward.

is no increase in intraocular pressure during accommodation. It is likely, however, that transient regulatory changes in production and outflow of aqueous take place during accommodation. A tonographic study is at present being conducted to test this possibility.

Because of the possibility that the system regulating the aqueous outflow is somehow involved in accommodation, we paid special attention to the trabecular zone in our studies.

The Schlemm canal region of the trabecular band showed no specific change. We observed neither blood filling of the canal nor emptying of blood, where blood was present, in connection with the act of accommodation. We did, however, note a subtle and interesting change in the whole trabecular zone. At first this change was confusing and difficult to define, but close study has led us to the following conclusions:

Whenever a change in adjustment of the eye occurred (either from distance to near fixation, or vice versa) the fine crisscross striation * of the trabecular zone, when seen, was blotted out, and it became homogeneous in appearance in about one-half the subjects. At the same time, its color underwent a slight change; it lost some of its gray character and became more whitish, or porcelain-like in appearance. The changes in the trabecular zone lasted only for a few seconds. The normal texture and the normal color were soon restored, but with each change in fixation the cycle began anew. In other cases where the texture was too fine for observation, only the value change was evident. The width of the trabecular band remained unchanged, except in one 15-year-old girl, in whom it was felt that there was a slight broadening. But it was difficult to estimate this change, just as in the case of the scleral spur, because of the initial movement of the image.

Two of the subjects presented portions of narrow anterior border-rings of Schwalbe. In a 15-year-old female subject the ring projected slightly toward the anterior chamber during relaxation. It was pulled nearer into alignment with the cornea and inner trabecular surface during accommodation. In both subjects the ring was more pearly-white in relaxation and grayer in color during accommodation.

The changes in the trabecular zone were so subtle that any printed illustration would require exaggeration, and therefore be misleading.

6. *Lens.*—The very central portion of the posterior lens surface was beyond the edge of the gonioscopic view. We were, however, astonished to find that in the young, normal eyes which we examined it was possible to see the posterior lens surface nearly to the

Fig. 4.—Apex of angle of anterior chamber.

A, during relaxation of accommodation. Relatively limited exposure of ciliary body band (*Cil. b. b.*) and root of iris; *S. sp.*, scleral spur; *Schw.*, anterior border-ring of Schwalbe.

B, during active accommodation. Increased exposure of ciliary body band and iris root, due to backward movement of last roll of the iris. A mild curvature in slit-beam section, indicating slight change of contour over ciliary body band, is shown at *a*.

* The subjects between the ages of 22 and 26 showed more meshwork texture than had been expected, and more than had been seen in younger normal subjects studied in other gonioscopic investigations.

equator without the use of mydriatic drugs both during relaxation and during accommodation. Under ideal conditions about three-fourths of its superior and inferior aspects could be studied. In a few subjects the view of the superior portion was more limited, while the inferior was well exposed.

In the region of the lens periphery, along the lines of insertion of the zonule, we noted that the lens capsule changed plane, so that the optical section of the posterior capsule showed a noticeable angulation at that point.

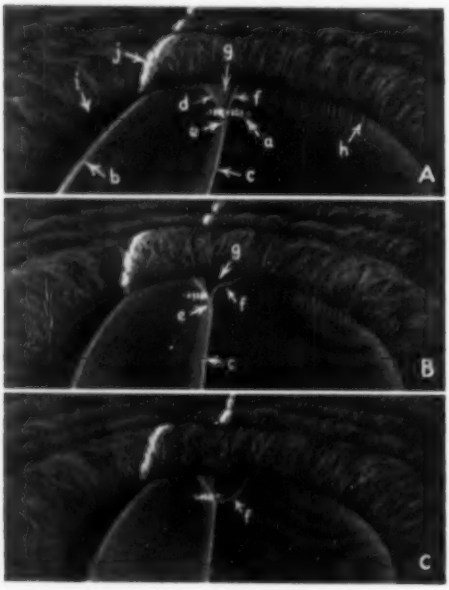


Fig. 5.—Changes in region of periphery of posterior lens surface.

A, during relaxation of accommodation. (*a*) Specular reflection of points of insertion of posterior zonular fibers; (*b*) slit-beam section of anterior lens surface; (*c*) posterior lens surface just central to ligamentum hyaloideocapsulare (Wieger); (*d*) posterior lens surface between latter line and equator; (*e*) point of angulation of lens surface; (*f*) surface of vitreous (posterior wall of canal of Petit); (*g*) space filled by faint flare, indicating zonular fibers; (*h*) zonular fibers seen by direct and indirect retroillumination; (*i*) pupillary margin; (*j*) slit-beam section of iris.

B, during active accommodation. (*c*) Peripheral posterior lens surface flattened; (*e*) angulation increased largely by flattening of latter; (*f*) anterior vitreous relaxed and bowed forward; (*g*) optically empty space in front of vitreous.

C, exceptional appearance in active accommodation. (*f*) Surface of vitreous along canal of Petit bowed backward.

Except in one 15-year-old female subject in whom this angulation was quite obtuse, at least during relaxation, the angle was rather acute in all instances (Fig. 5). This angulation is shown clearly in one figure of Goldmann's excellent monograph.[2] This figure also shows another relucent line, originating at or near the apex of the angulation. This line, which we could always observe when the view extended beyond the angulation in the lens capsule, passed backward and outward, away from the lens capsule. It is a slit-beam section of the anterior surface of the vitreous where it recedes from the lens at the so-called ligamentum hyaloideocapsulare of Wieger and forms the posterior wall of the canal of Petit. During relaxation of accommodation a grayish flare, representing the zonule of Zinn, was seen in the space between the optical section of the posterior lens surface and the face of the vitreous body.

The equator of the lens and some expanse of the anterior surface, as well as the anterior zonule and the ciliary body, were hidden by the iris.

The typical changes occurring in the lens in connection with accommodation are now described. All changes were consistent in direction, but they varied slightly in degree.

During relaxation of accommodation the slit-beam section of the posterior lens capsule was a rather flat, smooth curve, convex toward the vitreous body, up to the angulation previously described. The optical section of the face of the vitreous body recurved moderately a short distance beyond its separation from the line representing the posterior lens surface (Fig. 5*A*). The impression gained in all cases was that the vitreous surface was under slight tension during relaxation and that it rested against, or very near, another tissue. This latter impression was based upon the fact that the space between the vitreous face and the peripheral posterior lens capsule was filled with a faintly relucent substance.

During accommodation the slit-lamp section of the posterior lens capsule just central to the line representing the ligamentum hyloideocapsular flattened out. In two subjects

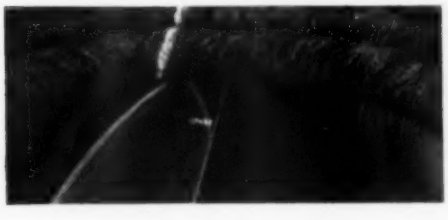
it even became slightly bowed in the opposite direction, giving the impression that the central part of the posterior lens capsule must have formed a Cupid's bow type of cross section.

At the same time in a few cases the optical section of the lens capsule beyond the point where it separated from that of the vitreous face tended to tilt slightly more forward than during relaxation. In the others it remained unchanged, but in either event the angle formed between the two capsular areas was slightly increased.

The most startling change came in the character and position of the face of the periphery of the vitreous. The slit-lamp section of this surface became markedly curved, and no longer appeared to be under tension. In all but two cases the curve was convex toward the zonule (Fig. 5*B*). In the two exceptional cases (Fig. 5*C*) the convexity was toward the vitreous body.

Whereas the space between the peripheral posterior lens capsule and the vitreous face was filled with a hazy grayness during relaxation (Fig. 5*A*), there was an optically empty space adjacent to the vitreous surface during accommodation (Fig. 5*B* and *C*). It would almost appear that aqueous from the posterior chamber had passed through the zonule into the canal of Petit. It so happens that one of the subjects having the exceptional appearance of the vitreous face shown in Figure 5*C* also had the most marked bowing backward of the central iris during accommodation. Such extreme movement might easily follow the free flow of aqueous out of the posterior chamber into the canal of Petit.

Fig. 6.—Same region of same eye as in Figure 5*C*, but under effect of cycloplegia, showing exaggeration of effect of normal relaxation.



One of the subjects showing the bowing backward of the optical section of the vitreous face was studied gonioscopically after administration of a cycloplegic. The resulting picture of the region of the canal of Petit (Fig. 6) was an exaggeration of that seen in normal relaxation. The surface of the vitreous periphery and the posterior lens capsule lay in the same, almost flat plane in the gonioscopic view.

CONCLUSIONS

Each of the various mechanical changes observed gonioscopically during accommodation were not seen equally well in all subjects. Nevertheless, the changes were sufficiently constant that one may state with confidence that they all appeared to bear out the Helmholtz theory of accommodation. There were the classical changes in the position of the iris and the lens and the redistribution of volumes within the anterior chamber. The impression was also gained that there was relaxation of tension by the zonule during accommodation, with recurving of the relaxed lens capsule to create greater curvature of the central lens surface. Finally, our new observations on the periphery of the vitreous surface strongly suggested that the vitreous body, far from pressing upon the periphery of the lens, was actually under reduced tension during accommodation.

SUMMARY

The classical changes in position and contour of the structures of the anterior segment of the eye during accommodation are enhanced by observation in slit-lamp gonioscopy.

Some new observations are reported. One relates to the trabecular zone, which presents subtle, though definite, changes in texture and color whenever a change in adjustment of the eye occurs. The other relates to the periphery of the lens, where the optical section of the posterior lens capsule shows that the angulation between the central and the peripheral part of the capsule becomes more acute during accommodation, owing to flattening or recurving of the portion just cen-

tral to the so-called ligamentum hyaloideocapsulare. A measurable change is observed in the slit-lamp section of the periphery of the vitreous, indicating a reduction of tension during accommodation. Lastly, an optically empty space is seen in the region of the canal of Petit during accommodation.

All observations tend to support the Helmholtz theory of accommodation.

Further studies of the phenomena observed in the trabecular zone are necessary. It is possible that they may throw light on the question as to whether accommodation plays a beneficial or a detrimental role, or no role at all, in eyes predisposed to glaucoma.

University Hospitals.

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The Management of Monocular Cataracts

EVERETT L. GOAR, M.D., Houston, Texas

For the purpose of this discussion a monocular cataract will be defined as advanced opacification of the crystalline lens in one eye, while the other lens is clear or has opacities that have not yet affected visual acuity. The case of the infant born with monocular cataract is another problem and will not be discussed here.

Until recently a cataract in one eye was allowed to go to maturity and beyond as long as the vision remained useful in the other eye. An ancient dictum said one should wait until vision dropped to 20/70 in the better eye before operating on the other. The reasoning behind this was that as long as the patient could see well enough to get around and to do close work with the better eye, the eye operated on would be of little or no good. A patient could not wear a cataract lens before the eye operated on and a correcting lens before the one not operated on because of the difference in size images. That this opinion is still held, as well as the belief that if a good eye is obtained by surgery the other had best not be operated on, is evident from an article that appeared in 1953.[1] This sort of teaching does not take into account what happens to the patient when this old principle is followed, and, in the light of modern methods, it is, in my opinion, indefensible.

Let us follow the patient who loses binocular vision when he has a cataract far advanced in one eye with little or none in the other. First occurs loss of convergence, soon followed by exotropia, and occasionally a vertical deviation. There is no way to measure exotropia for distance with an eye blind from cataract, but one can be sure if the convergence near point is becoming remote, exotropia is on the way. If this is allowed to become permanent and of high degree, it will be extremely difficult to secure fusion later, although Constantine and McLean * have accomplished it by the use of prisms whose strength is gradually reduced. However, it is better to operate before this unpleasant complication occurs. I have found that daily forced convergence exercises with a pocket flashlight will prevent exotropia if they are started before convergence is completely lost and the instructions are followed faithfully. This exercise is for persons who want to delay operation or are satisfied with their present status of seeing with one eye. The other complication that may occur is secondary glaucoma due to intumescent or to hypermature cataract. The clinical picture is somewhat different in the two conditions, but both are serious. In the former, the lens is swollen, the anterior chamber shallow, and all the other signs and symptoms of acute congestive glaucoma are evident. In the latter, the anterior chamber may be of normal depth or deeper than normal, the lens may be shrunken, the cornea steamy, and if Descemet's membrane is visible with the biomicroscope, there are cellular deposits. This is a frequent complication of hypermature cataract, and, according to the Irvines,[2] is due to blocking of the angle with macrophages, which are formed as a result of lens material seeping through the thinned posterior capsule. The tension in this type of glaucoma can usually be reduced by miotics

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From the Department of Ophthalmology, Baylor University College of Medicine.

* Personal communication to the author.

for a while, but it is necessary to remove the lens soon before it rises again. I once removed the lens in capsule in the condition just described, and on the first slit-lamp examination, 10 days later, saw what appeared to be lens fibers lying between the iris and the hyaloid, visible through the coloboma.

Mr. Ridley, of London, has attempted to solve the problem of one-eyed cataracts by use of an acrylic implant, and he reports considerable success. Only a few American ophthalmologists have had the temerity to do this operation, and, except for one who has had considerable experience with the method, most have abandoned it. The acrylic implant is still on trial, and no one knows what will happen to these eyes 10 years after operation.

There is a much simpler and, I believe, safer way of managing the one-eyed cataract than by putting what amounts to a foreign body inside the eye—that is, by putting it on the outside. I refer to the use of a contact lens. It would seem superfluous to write on this were it not for the fact that but one article on the subject has appeared in the American literature,[8] and that in a class of about 60 ophthalmologists at the last meeting of the American Academy of Ophthalmology and Otolaryngology only 4 had any experience whatever with this method of treating monocular aphakia.

My first use of a contact lens in monocular aphakia was over five years ago. My first two patients were middle-aged persons—one a bookkeeper, the other a housewife. Both were fitted with scleral contact lenses. The bookkeeper wears his all day and is so happy with it that I have not tried to change it to a corneal lens. The housewife wore her contact lens two years, but after the second eye was operated on she went to regular cataract lenses.

About this time the Touhy corneal lens became available and, with two exceptions, has been used in all my cases. The exceptions were not satisfactory. The great fault with the Touhy lens is that it is easily lost, and in prominent eyes it has a tendency to fall out. They must be fitted with extreme care, and a busy ophthalmologist does not have time for this. It is a job for a skilled and patient technician. Parenthetically, the doctor needs considerable patience also, for these persons wearing a contact lens need more time spent with them than those who wear conventional cataract lenses, and they are likely to drop in any time without appointment.

The procedure I have used in monocular aphakia is as follows: The eye operated on is refracted 8 or 10 weeks after operation. If the vision is not 20/30 or better, the examination for contact lens stops here. If the vision is good, the prescription with vertex distance is sent to the technician. He tries the patient out for tolerance and teaches him to insert and remove a lens from a trial set. If the patient is too clumsy, too nervous, or too impatient, he is not a good subject for a contact lens. The corneal sensitivity in an aphakic eye seems to be diminished, and the tolerance for the lens is much greater than in the nonaphakic eye. If the patient seems a potential wearer, the technician takes his own measurements and orders the lens. At the same time, the patient deposits a sum which includes the price of the lens, insurance against loss, and the technician's fee. If he cannot wear the lens satisfactorily, he returns it within a few weeks and the money is refunded. This can be done because the lenses are molded and can be returned to the factory. When the lens arrives, the patient is instructed to wear it a half-hour and increase the time a half-hour daily. When he can wear it comfortably four or five hours, he returns to the ophthalmologist. If his vision is 20/30 or better, and if the lens fits well and is comfortable, he is to keep this one. Vision can often be improved by adding a weak convex lens to his spectacle lens. However, if he needs much addition for good vision, another lens is ordered. At this time the Worth dot test is made for near and distance. If the patient does this test accurately, he has primary fusion. If he has diplopia, he has muscle imbalance, and the type and amount are determined by the screen

test. Not a few of these patients have a vertical imbalance, and it is necessary to incorporate prisms in their glasses. If the patient has primary fusion, that is all he needs to be a successful contact-lens wearer. If he has not, there is some question about it, because he may have an imbalance that cannot be overcome by prisms or training. I have considered operating on the patients who are exotropic, but have not done so yet. At this visit the contact lens is removed, and the cornea is stained with fluorescein and examined with the slit lamp. If the lens has been in several hours and there are a few flecks in the epithelium, they need cause no concern. However, if there is enough stippling to cause reduction in vision, the lens is too tight and is not allowing the tears to enter properly. It must be adjusted or changed. I have never seen an irreversible change or corneal damage, though some patients wear the lens 18 hours a day without removing it. One woman who had two contact lenses took a nap with one in. When she awoke, being unable to see, she put the other in. The first one had slipped over on the temporal sclera and remained there 72 hours. She came complaining of the eye being irritated and I removed the lens, to her astonishment.

The reason a person can wear a contact lens comfortably and fuse the image with that of the other eye is that there is not the huge difference in size in the two images such as exists with a spectacle lens before the aphakic eye. As the convex lens moves toward the nodal point of the eye, the image diminishes in size. Linkz measured several aphakics on the eikonometer for McLean and Constantine and found an average size difference with a contact lens of 7.5%. It is estimated that a cataract lens set at the anterior focal plane of the aphakic eye causes an increase of 30%.

The grade of fusion does not seem to make much difference. Constantine and McLean found that almost half their contact-lens wearers had third-grade fusion. I have not measured them on the amblyoscope, but many patients can do bar reading successfully, and some will read well down on the Keystone sterometric card.

I have 35 patients who have worn contact lenses happily and have continued to lead active lives with them. The second patient mentioned who had a scleral contact lens gave it up after her second eye was operated on. Only two other wearers have come to the second operation, and these patients wear the lenses in both eyes. Five patients who were fitted early in my experience gave them up, but one of these when fitted with a corneal lens now wears it. The ages range from 15 (traumatic) to 76, with an average of 55. Most of the contact-lens users keep the lens in from the time they arise until bedtime. Many have been advised to take the lens out at noon and leave it out an hour or two, but after a few months most wearers fail to follow this suggestion. It really seems to make no difference, as the cornea remains normal.

The persons who are to wear a contact lens must be selected with some care. Extremely nervous or very impatient persons are not very good candidates. Old and inactive persons should not have further burdens added to their lives. This form of therapy is reserved for those who are still in the active phase of life. Date of birth is not always the distinguishing factor. Some persons are too clumsy, or have a tremor, or are not intelligent enough to understand the purpose of the procedure. If the wearers are properly selected and are fitted by a skilled technician, they are the happiest of all cataract patients, because they have the vision restored in a blind eye, they do not have diplopia, and they have a field of vision almost normal in extent.

SUMMARY

The old belief that a cataract should not be operated upon as long as vision in the opposite eye is good should be discarded.

Mature and hypermature cataracts are more difficult to remove in capsule than if removed earlier. They should be allowed to reach this stage only in poor operative risks.

Complications are prone to occur in hypermature cataracts. The commonest are exotropia and secondary glaucoma.

Intelligent persons in the active phase of life, who are operated upon before exotropia occurs, will accustom themselves readily to wearing a contact lens, will get sufficient fusion to give them binocular single vision, and will enjoy a full field of vision on the side of the eye operated on. Some will continue to wear the contact lens in both eyes after both cataracts are removed because of the restricted field of vision and the annular scotoma produced by regular cataract lenses.

Monocular aphakics who wear a contact lens are the most satisfied of all cataract patients.

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The Ocular Significance of Intracranial Calcium Deposits

JOSEPH E. ALFANO, M.D.
and
HARVEY WHITE, M.D., Chicago

The importance of x-rays of the skull as a diagnostic aid in ophthalmological problems is now well established. These x-rays may be reported as normal except for the presence of calcium deposits in the substance or on the surface of the brain. In some cases these calcific deposits are considered as normal findings, whereas at other times they are of very definite ocular significance.

Calcification of the pineal gland (Figs. 1 and 2), calcium deposits in the choroid plexus of the lateral ventricle (Figs. 3-6), and osseous plaques in the falx cerebri (Fig. 7) may appear as normal findings. Calcifications of the Pacchionian granulations and calcifications in the hypophysis, although less common, may also be present normally.

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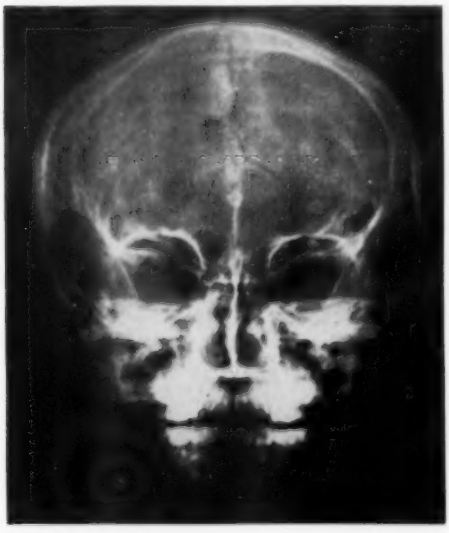
From the Departments of Ophthalmology and Radiology, Children's Memorial Hospital and the Northwestern University Medical School.

Fig. 1.—Calcified normal pineal gland, A-P view.



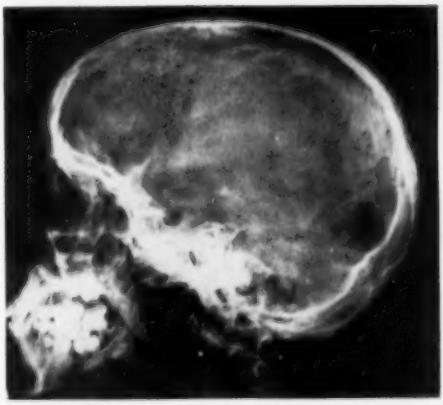


Fig. 2.—Calcified normal pineal gland, lateral view.

Pathologically, intracranial calcifications may be seen in toxoplasmosis; Sturge-Weber syndrome; tuberous sclerosis; intracranial arterial aneurysms [1]; slow-growing tumors, such as gliomas and meningiomas,[2] craniopharyngioma, and pituitary adenoma [3]; hypoparathyroidism [3]; arteriovenous malformations [1]; subdural hematoma,[4] and parasitic conditions.[5]

Miscellaneous conditions in which calcifications are sometimes found include encephalitis,[3] lead intoxication,[3] extreme old age,[6] amaurotic idiocy,[5] epilepsy,[6] chordomas,[4] cholesteatomas,[4] ependymoma,[5] and old intracranial abscesses,[4] tuberculomas,[4] and hematomas.[4]

In some cases, dense calculi are found in the substance of the brain for which no definite cause may be found.[5]

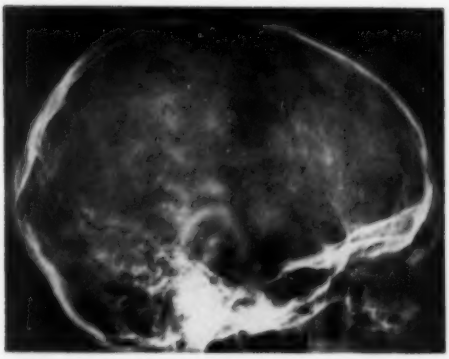


Fig. 3.—Calcifications in choroid plexus, lateral view.

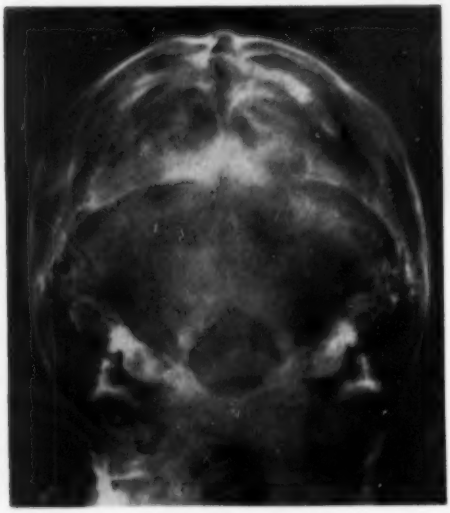
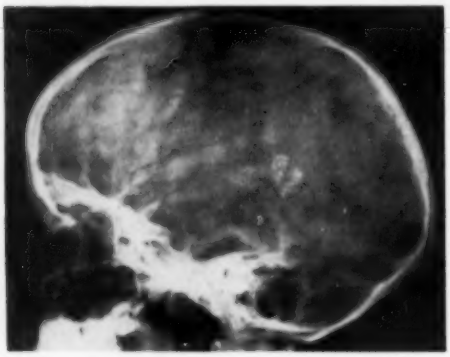


Fig. 4.—Calcifications in choroidal plexus, A-P view.

Fig. 5.—Isolated calcification in choroid plexus, lateral view.



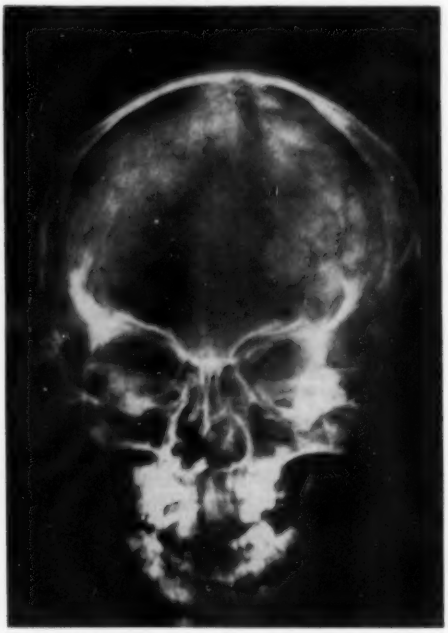


Fig. 6.—Isolated calcification in choroid plexus, A-P view.

Pineal Gland

Calcification of the pineal gland is thought to be present in approximately 50% of normal persons [6] (Fig. 1). Calcification of the gland is less common below the age of 20

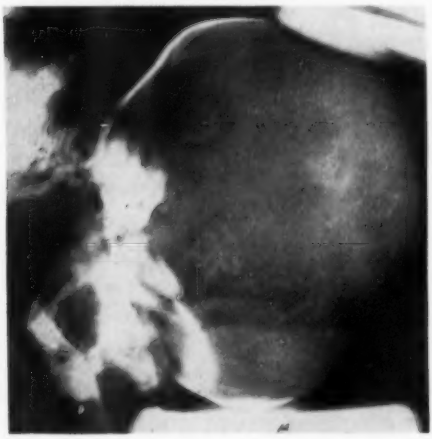
Fig. 7.—Calcification in falx cerebri, A-P view.

Fig. 8.—Shift of pineal gland to the right in a patient with a left parietal meningioma.

than in later years.[6] Pineal gland calcifications are significant only insofar as their displacement may indicate tumors of the brain or unilateral brain atrophy. With frontal and temporal lobe tumors (Fig. 8) the pineal gland is pushed backward; with parietal tumors,* downward, and with occipital tumors, forward.

* Case 1.

Fig. 9.—Spotty, disseminated calcifications in case of toxoplasmosis, lateral view.



PATHOLOGIC INTRACRANIAL CALCIFICATIONS

Toxoplasmosis

The congenital or early infantile variety of toxoplasmosis, which is probably of intrauterine origin, is characterized by Toxoplasma encephalitis, and almost invariably chorioretinitis.[7] The chorioretinal lesions are usually bilateral and frequently involve the macular area, leaving a typical sharply defined, punched-out scar, in the center of which exposed sclera may be seen and around which profuse irregular deposition of pigment may

Fig. 10.—A-P view, showing spotty calcifications, in same case as that in Figure 9.

be present. Single or multiple isolated patches of chorioretinitis may be present in the periphery of the retina.

Evidence of generalized and irregular involvement of the central nervous system includes head retraction, facial palsy, conjugate ocular deviations, strabismus nystagmus, absence of pupillary and vestibular reflexes, hydrocephalus, convulsions, and mental retardation.

In a large percentage of these cases disseminated deposits of calcium can be demonstrated on x-ray examination of the skull.

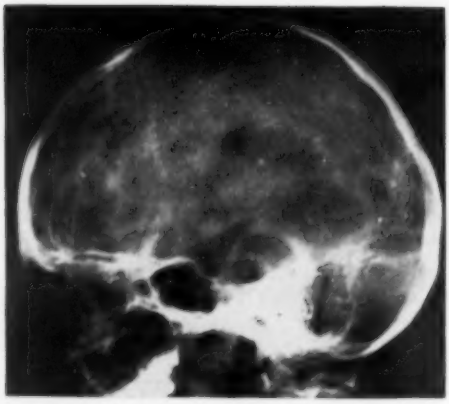


Fig. 11.—Small, spotty calcifications in toxoplasmosis, lateral view.

These calcifications are usually present at birth, are spotty, and are generally seen in the occipital region and/or disseminated throughout the substance of the brain (Figs. 9-12).†

Tuberous Sclerosis

The essential features of the complete syndrome of tuberous sclerosis (Bourneville's disease) are epilepsy, mental symptoms, neurologic symptoms, skin changes, and changes in the fundus.[8]

Epilepsy usually makes its appearance in the first two years of life and is present in most of the cases. The mental symptoms may vary from a mild degree of feeblemindedness to imbecility and idiocy. The neurologic symptoms are not very marked and at times may be absent. Defects which have been observed include hemihypertrophy, high palate, epicanthus, supernumerary digits, syndactyly, spina bifida, hydrocephalus, and cranial anomalies. Papilledema does not occur except in the rare case in which there is a growing glial tumor in the brain.

† Cases 1, 2, and 3.

Fig. 12.—Calcifications in A-P view in same case as that in Figure 11.

Fig. 13.—Multiple, irregular, spotty calcifications throughout substance of brain in case of tuberous sclerosis, lateral view.

The most important changes in the skin are known as adenoma sebaceum, which consists of a nodular rash on the face, distributed on the nasolabial fold and spreading upward on the cheeks and on the nose in butterfly fashion; changes in the pigment of the skin include café-au-lait spots, diffuse bronzing, vitiligo, and leucoderma.

Fundus changes in tuberous sclerosis are not common. Nodular, discrete, whitish-gray, mulberry-like masses, roughly about the size of the optic disc, may be found. They are usually located near the disc but may occur in the retinal periphery as well. Oval or circular grayish areas may also be found.

Fig. 14.—Calcifications in A-P view in same case as that in Figure 13.

An accompanying bilateral papilledema indicates the presence of cerebral tumors with a raised intracranial pressure.

In some cases cerebral calcifications may be present. The calcifications may be present at birth but are more likely to appear after the fifth year of life. They resemble the calcifications seen in toxoplasmosis (Figs. 13-15) ‡ in that they are spotty and disseminated throughout the brain substance. The calcifications in tuberous sclerosis are usually larger than those seen in toxoplasmosis.

Sturge-Weber Disease

The Sturge-Weber syndrome (Parkes-Weber-Dimitri disease, encephalotrigeminal angiomatosis) is characterized by vascular nevi occurring along the course of the trigeminal nerve and meninges, by Jacksonian convulsions, and by intracranial calcifications.[5] In some cases glaucoma occurs in the ipsilateral eye. The facial nevi are of the "port-wine stain" type and are situated especially over the forehead and eyelids. They are usually unilateral and on the same side as the cerebral lesion. Convulsions are a cardinal feature of this condition, and as a rule they are focal and contralateral to the nevus. Mental deficiency is present in many cases.

A choroidal angioma, sometimes associated with tortuous retinal vessels, and quite frequently with structural anomalies, may give rise to unilateral glaucoma on the ipsilateral side.

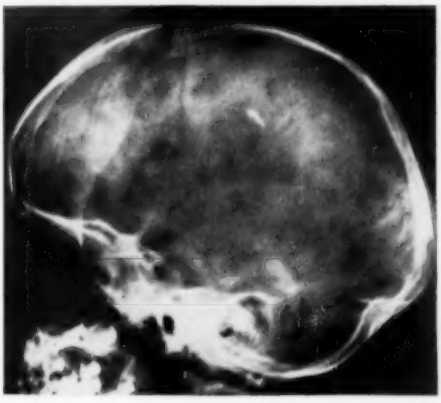
Cerebral calcifications are frequently observed. Although the calcifications may be present at any time after birth, they are more likely to appear after puberty. The calcifications § usually begin, and are most frequently present, in the occipital area, from whence they may extend forward to involve the parietal and frontal cortex. The calcifications usually appear on the surface of the cortex rather than in the substance of the brain and produce a highly characteristic roentgenologic appearance, resembling multiple worms. These areas consist of tortuous parallel lines [6] surrounded by fine, calcific stippling (Fig. 16).‖ Histologic examination [6] reveals them to be calcified telangiectases in the cerebral cortex. Arteriography is usually negative.

‡ Cases 4 and 5.

§ Buchanan, D.: Personal communication to the authors.

‖ Case 6.

Fig. 15.—Spotty, disseminated cerebral calcifications in patient with tuberous sclerosis, lateral view.



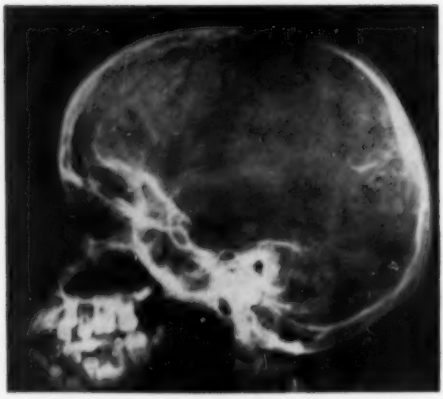


Fig. 16.—Worm-like calcifications in postparietal region in patient with Parkes-Weber-Dimitri syndrome.

Congenital Vascular Malformations

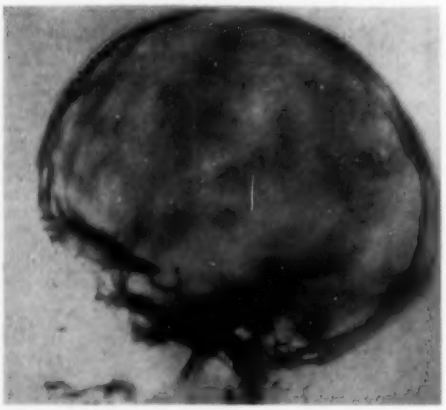
The von Hippel-Lindau disease is distinguished by the fact that it does not show cerebral calcifications.

Congenital cerebral arteriovenous communications, which are in reality persistent embryonic vascular plexuses, occasionally calcify. These calcifications appear as rings or fine spiral streaks in the region of the aneurysm (Fig. 17). Angiography may establish the diagnosis.

Intracranial Tumors

All the slow-growing intracranial tumors may calcify, in particular gliomas and meningiomas.

Fig. 17.—Spiral calcification in vascular malformation in occipital area.



Gliomas.—Gliomas constitute the commonest intracranial tumors, forming about 50%. They occur more frequently in the cerebrum than in the brain stem or the cerebellum and are as often benign as malignant. Lindblom ¶ observed calcification in 5% of all gliomas (Fig. 18).#

Meningioma.—Meningiomas constitute the second most frequent group of intracranial tumors, forming about 17%. They are located most commonly in the convexity of the cerebrum, where they occupy a sagittal position in relation to the superior sagittal sinus. They are seen less often in the region of the olfactory grooves, the tuberculum sellae, the lesser wing of the sphenoid bone, and the apex of the petrous portion of the temporal bone.

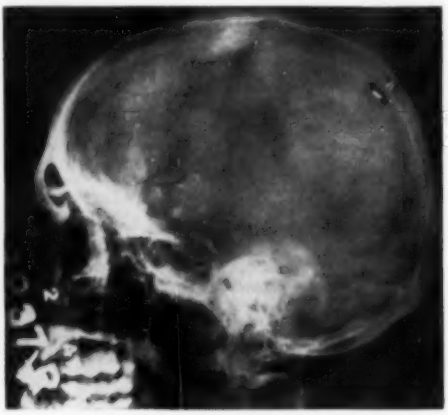


Fig. 18.—Calcified glioma (astrocytoma) of right temporal lobe.

Calcification [6] in a meningioma is, however, not too common, occurring less frequently than in gliomas. Since calcification [6] of the dura mater is occasionally observed after trauma, such a condition must not be confused with a calcified meningioma.

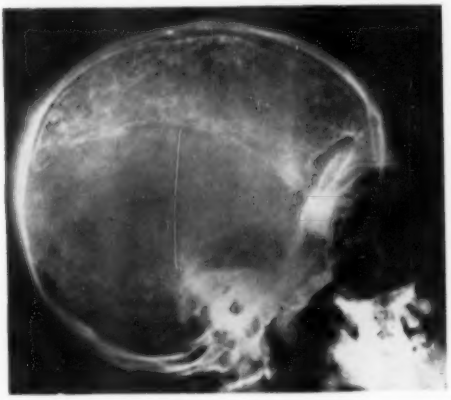
Miscellaneous Tumors.—Tuberculomas,[7] old subdural hematomas (Figs. 19-20 *), and healed brain abscesses [2] may show dense calcification.

¶ Cited by Case.[6]
# Case 7.
* Case 8.

Fig. 19.—Calcified plaque in meninges in a 12-year-old boy resulting from subarachnoid hemorrhage at the time of birth, A-P view.

Martin and Lemmen [7] studied 1577 verified tumors of the central nervous system. Of these 1577 tumors, 207, or 13.1%, showed calcification microscopically. Roentgenologic study revealed that of these 207 tumors with microscopic evidence of calcification, 54.7% showed evidence of calcification on x-ray examination. Patients who had evidence of calcification experienced significantly longer periods of survival.

Fig. 20.—Lateral view in same case as that in Figure 19.



Craniopharyngioma and Pituitary Adenoma

Craniopharyngioma are congenital tumors, usually cystic in nature, which arise from embryonic remnants of the craniopharyngeal duct. Symptoms usually appear between the 10th and the 25th year, and in a typical case constitutional signs of hypopituitarism, chiasmal defects, and radiographic evidence of erosion of the clinoid processes are present.

According to Ford,[5] it is estimated that calcifications are found in more than 75% of these tumors. These calcifications may appear as small flecks, curved linear shadows, or enormous calcareous masses lying in the suprasellar area (Fig. 21 †). Case [6] feels that in the absence of these calcifications

Fig. 21.—Suprasellar calcification in craniopharyngioma.

these tumors cannot be diagnosed roentgenologically. While suprasellar calcifications are almost pathognomonic of a craniopharyngioma, other conditions, such as gliomas of the optic chiasm [4] and hypothalamus,[4] suprasellar meningiomas,[4] chordomas,[4] internal carotid artery aneurysms,[4] and old hematomas,[4] may produce similar calcifications in the suprasellar area. The calcifications of an optic chiasm glioma or a glioma of the hypothalamus tend to display a nodular configuration. Although a suprasellar meningioma rarely shows calcification, when they do occur, these calcifications appear as dense and homogeneous densities anteriorly, above the

† Case 9.

Fig. 22.—Suprasellar curved-linear calcification in saccular supraclinoid aneurysm of internal carotid artery.

sphenoidal plane. Calcified chordomas in this region produce a dense calcification that destroys the dorsum sellae, the clivus, and the apex of the petrous portion of the temporal bone.

Calcifications may occur in a normal pituitary gland, and it is estimated that calcifications may be seen in 3% of pituitary adenomas.[4] If the calcified adenoma is sufficiently large to extend beyond the confines of the sella turcica, the calcified shadow may be seen in the suprasellar region. Suprasellar calcifications produced by internal carotid artery aneurysms will be discussed in the next section.

Intracranial Artery Aneurysms

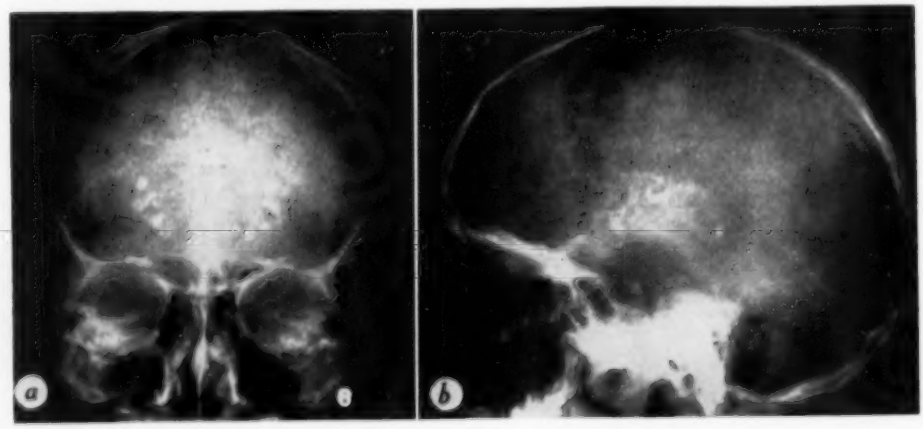
Internal Carotid Artery.—Aneurysms of the internal carotid artery may be supraclinoid or infraclinoid in the cavernous sinus.

Supraclinoid aneurysms may be of the small berry type, occurring at the junction of the internal carotid with its branches; larger saccular aneurysms of the carotid before it gives off its branches, and diffuse, usually cylindrical, dilatations of the intracranial portion of the carotid trunk. Aneurysms of the infraclinoid portion of the carotid (cavernous sinus) are usually saccular in type. It is thought that approximately 15%[1] of all aneurysms of the internal carotid show calcific deposits.

Internal carotid aneurysms in the cavernous sinus will only occasionally show evidence of calcification.

These calcifications mean deposition of calcium in defective walls and indicate a long duration of the aneurysmal defect. On radiographic examination these calcifications may appear as small circular shadows, irregular masses, or diffuse, scattered areas, above the sella or in the carotid canal on one or

Fig. 23.—Characteristic appearance of symmetrical calcifications of cerebral basal ganglia (spontaneous parathyroid insufficiency). (*a*) Bilateral symmetrical distribution of shadows of calcification; (*b*) extent of shadows of calcification. Reproduced from Camp, J. D.: Radiology **49**:568, 1947; published by the Radiological Society of North America, Syracuse, N. Y.



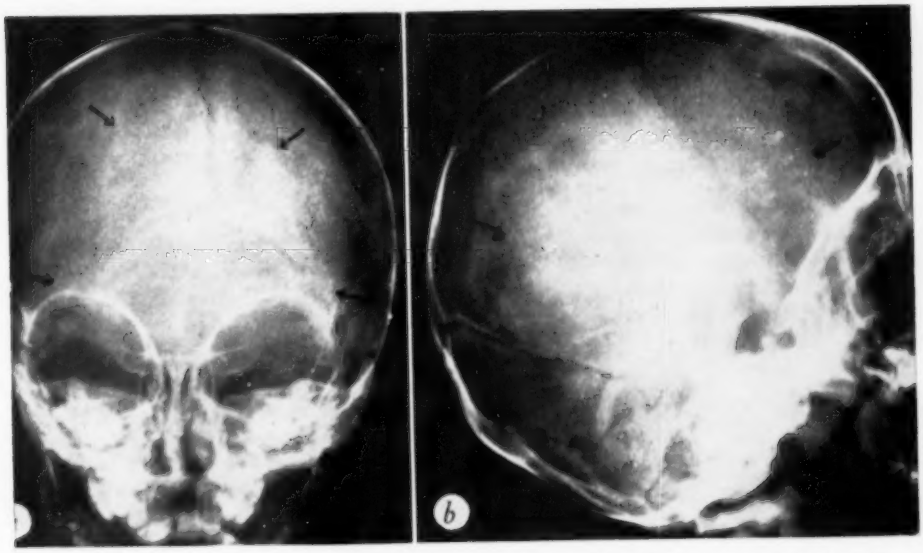


Fig. 24.—Symmetrical calcification of cerebral basal ganglia and extensive calcification in cerebrum in a case of encephalitis in infancy. Reproduced from Camp, J. D.: Radiology **49**:568, 1947; published by the Radiological Society of North America, Syracuse, N. Y.

both sides, or as linear shadows, which are usually curved (Fig. 22).‡

Vertebral and Basilar Arteries.—Calcifications in these vessels [1] are difficult to detect. The roentgenographic view is not so clear because of the interposed petrous process and the mastoid bone.

Hypoparathyroidism

Idiopathic hypoparathyroidism, which is probably dependent upon intrinsic factors in the parathyroid gland, is characterized [3] by a low serum calcium, a high serum inorganic phosphorus, absence of renal insufficiency, and chronic tetany.

This condition is replete with signs of ophthalmologic interest,[8] including conjunctivitis, keratitis, blepharospasm, photophobia, cataract, papilledema, and optic neuritis.

Roentgenologically bilateral and symmetrical calcifications have been observed in the basal ganglia § (Fig. 23). However, calcifications of this type, while characteristic

‡ Case 10.

§ References 3 and 11.

Fig. 25.—Calcified areas scattered through the more superficial portions of the cerebrum in a case of epilepsy. Reproduced from Geyelin, H. R., and Penfield, W.: Arch. Neurol. & Psychiat. **21**:1020, 1929.

of hypoparathyroidism, are not specific. These findings are sometimes observed in extreme old age,[6] amaurotic idiocy,‖ encephalitis ‖ (Fig. 24), lead intoxication,[3] and epilepsy ¶ (Fig. 25).

Parasitic Conditions

While the occurrence of ocular conditions produced by parasites is not common, the question of a parasitic etiology in a pseudoglioma in a child occasionally presents itself. It is well established that ocular conditions can be caused by parasites.[10] Cysticerci and Ecchinococcus cysts are sometimes calcified and give the appearance of numerous small calcified areas scattered throughout the brain. Any such findings should lead to a suspicion of parasitic disease.[5]

Undetermined Causes

In some cases, dense calculi are found in the substance of the brain for which no definite cause may be found.[5] As a rule, they are multiple and vary in size from a few millimeters to several centimeters.

DIFFERENTIAL DIAGNOSIS

Intracranial calcifications must be distinguished from ocular, intraorbital, and intracanalicular depositions of calcium.

Ocular Conditions

Calcium deposits in the cornea, in the form of a band-shaped keratopathy, may be occasionally seen on routine x-ray examination of the skull. While most forms of cataract show no evidence of calcium deposits on routine skull x-rays, a calcified lens can produce a characteristic, unmistakable shadow.

In a large proportion of cases of retinoblastoma small islets of calcium are present in the tumor, and it is estimated that in 75% of cases these deposits may be seen radiographically.[10] Although calcification may occur in conditions listed as pseudoglioma, such as retrolental fibroplasia, and in inflammatory conditions producing organized inflammatory tissue in the vitreous, such as tuberculoma, gumma, exudative retinitis of Coats, metastatic ophthalmitis, detached retina, angiomata, and cysticercosis, it is rare for calcareous degeneration to be present in the eye of a child in circumstances other than a retinoblastoma.[10]

Orbital Conditions

Calcium deposits may be present in a retinoblastoma that has extended into the orbit. Calcium may be deposited in an orbital hematoma or abscess or in a glioma of the intraorbital portion of the optic nerve. Hemangioma # or varix ** of the orbit may occasionally become calcified.

Intracanalicular Depositions

Intracanalicular deposits of calcium may follow fracture of the optic canal in which callus formation has taken place in the repair of the fracture, and in a calcified glioma of the optic chiasm that has extended into the optic canal.

SUMMARY

Radiologic examination of the skull for evidence of intracranial calcifications may serve as a valuable diagnostic aid.

Calcifications may be seen normally in the pineal gland and choroid plexus of the lateral ventricles and as osseous plaques in the falx cerebri. Less commonly, calcifications may occur normally in the Pacchionian granulations and in the hypophysis.

The conditions of prime ophthalmologic importance in which intracranial calcifications may be seen include toxoplasmosis, Sturge-Weber syndrome, tuberous sclerosis, intracranial arterial aneurysms, craniopharyngioma, pituitary adenoma, slow-growing gliomas and meningiomas, hypoparathyroidism, subdural hematoma, cerebral arteriovenous malformations, and parasitic conditions.

Intracranial calcifications may also be seen in such conditions as encephalitis, lead intoxication, extreme old age, amaurotic idiocy, epilepsy, and old intracranial abscesses, tuberculomas, and hematomas.

‖ References 3 and 6.

¶ References 6 and 12.

References 9 and 13.

REPORT OF CASES

CASE 1.—A 55-year-old white man gave a history of weakness in the right leg of 14 months' duration, jerking movements of the right foot and numbness of the right side of the body for 1 year, weakness of the right arm for 1 year; inability to concentrate on his work for the past 11 months; left-sided headaches of 8 months' duration; a "loss of words" for about 2 months, and difficulty in writing for about 1 month. One month previous to admission he had had a generalized convulsion.

Physical examination was noncontributory, except for a right-sided hemiparesis and a positive Babinski on the right side. The eyes were normal in all respects, as were the visual fields. Roentgen examination of the skull revealed a shift of the pineal gland, which was calcified, to the right, as shown in Figure 8. A ventriculogram revealed a shift of the ventricles to the right.

At operation a meningioma of the left parietal region was removed.

Pathologic examination revealed that the tumor was richly vascular. The cells were arranged in large and small whorls. There were gently streaming masses, with in some places a great deal of fibrous stroma and in others the appearance of almost pure fibroblasts. There were no areas of necrosis.

CASE 2.—A white male infant was first seen at the age of 1½ days. The infant was delivered after a labor of three and a half hours. He was a breech presentation and was delivered by forceps. The mother gave no history of prenatal illnesses. At about 13 hours after birth it was noticed that the infant's temperature was 94 F; there were twitchings of the arms and legs, and the child was rigid. He was also jaundiced, and there were multiple petechiae in the skin.

Examination at this time revealed an acutely ill infant who was having clonic and tonic contractions of all extremities. Reflexes were all hyperactive. The skin was jaundiced, with scattered petechial hemorrhages. The fontanelles were bulging. The abdomen was distended, and the liver and spleen were palpable. Examination of the eyes revealed a marked nystagmus. The left eye was smaller than the right. The sclerae were icteric, and there was esotropia of the left eye. There was a bilateral macular and disseminated chorioretinitis.

Both the mother and the patient showed a positive complement fixation dye test for toxoplasmosis in a dilution of 1:4096. Organisms resembling Toxoplasma were found in both the urine and the spinal fluid. Thirty mice were injected with the cerebrospinal fluid. Ten were injected intracranially, and all died in from 1 to 20 days. Twenty were injected intraperitoneally, 10 of which died. Skull x-rays revealed calcifications, as shown in Figures 9 and 10. The child became progressively worse and died on the 14th day. Pathologic examination of the brain revealed hydrocephalus and multiple disseminated calcifications. The brain was completely abnormal and showed severe destruction of both hemispheres. The lesion extended through and ended in the midbrain. The upper part of the spinal cord and the pons and cerebellum appeared normal; however, in the large extension of the cerebellum and in both hemispheres there is a moderate degree of subarachnoid hemorrhage.

Sections from three blocks of the brain were available for microscopic study, as shown in Figures 26-28. The first (Fig. 26) is derived from the cerebellum. The chief abnormality noted is marked thickening of the pia-arachnoid by intact and partially degenerated red blood cells, large and small round cells, and occasional polymorphonuclear leucocytes.

Sections derived from the second block (Fig. 27) contain portions of choroid plexus. The surrounding tissue is almost totally replaced by an infiltration of large and small round cells. These cells tended to center about foci of calcification.

Sections derived from the third block (Fig. 28) are from the cerebral cortex. The meninges are involved by changes similar to those noted in the cerebellum. Also present in these sections are accumulations of large and small round cells and occasional polymorphonuclear leucocytes centering about foci of fine granular calcification. Toxoplasma organisms were identified in these lesions.

CASE 3.—An 11-year-old white boy was admitted to the hospital because of a low-grade fever and frequent nosebleeds. He gave a history of poor vision since birth and frequent falling-out spells. Physical examination was noncontributory. The best visual acuity was 20/100 in the right eye and counting fingers at 2 ft. in the left eye. There was bilateral macular chorioretinitis. Skull x-rays revealed calcifications, as shown in Figures 11 and 12. There was no hydrocephalus, and the intelligence quotient was 88.

CASE 4.—A 3-year-old white boy was admitted to the hospital because of left-sided convulsions, of about one and one-half years' duration. Physical examination, including a neurologic study, was noncontributory except for some "white spots" in the skin. The patient had had one brother who suffered from convulsions and died at the age of 10 years. Skull x-rays revealed calcifications, as shown in Figures 13 and 14.

CASE 5.—A 3-year-old white boy had a history of convulsions of one year's duration. He was mentally alert, and there was no physical findings except for adenoma sebaceum, characteristically distributed over the face. Skull x-rays revealed calci-

Fig. 26.—Microscopic section from cerebellum, showing thickening of pia-arachnoid by intact and partially degenerated red blood cells, large and small round cells, and occasional polymorphonuclear leucocytes (toxoplasmosis).

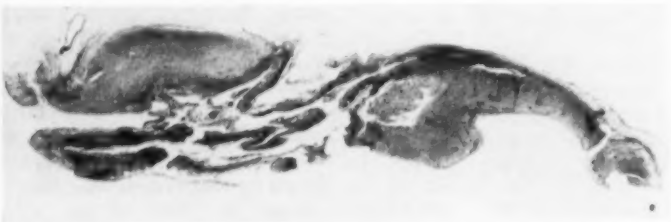


Fig. 27.—Microscopic section from choroid plexus, showing almost total replacement by an infiltration of large and small round cells, which tend to center around foci of calcification (toxoplasmosis).

Fig. 28.—Microscopic section from cerebral cortex, showing involvement of the meninges by changes similar to those in the cerebellum. There were also accumulations of large and small round cells and occasional polymorphonuclear leucocytes centering about foci of fine granular calcification. Toxoplasma organisms were identified in these lesions.

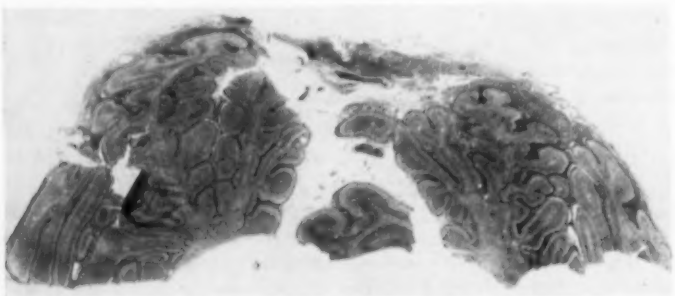


Fig. 29.—Front view of patient, showing hemangiomatous involvement of skin of left eyelid, forehead, nose, cheek, and lips.

Fig. 30.—Lateral view of patient shown in Figure 29.

fication, as shown in Figure 15. The patient has an older sister with tuberous sclerosis.

Case 6.—A 9-year-old white girl was seen because of frequent nosebleeds and several birthmarks on the face. Physical examination, including a neurologic study, was noncontributory except for a hemangioma of the left side of the face, extending up on the scalp to the vertex, back to the occipital region, anterior to the ear, down to the level of the mouth, and on to the nose. This could also be seen on the roof of the mouth (Figs. 29-31). The frequent nosebleeds were the result of angiomatous polyps. The best result of visual acuity with correction was 20/100 in each eye. There was bilateral ocular nystagmus. The right eye was otherwise normal. In the left eye the cornea was clear, and appeared to be larger than the right. Ocular tension, however, was normal in each eye. The left disc was normal, but the retinal vessels appeared dilated and tortuous. There was an alternating divergent strabismus of 35 degrees. Skull x-rays revealed calcifications, as shown in Figure 16.

Fig. 31.—Involvement of roof of mouth in case shown in Figures 29 and 30.

Case 7.—A 30-year-old white man gave a history of generalized headaches of 10 years' duration, with generalized convulsive seizures for five and a half years preceding this hospital admission. Physical examination was noncontributory. Skull x-rays revealed a calcified tumor mass overlying the pos-

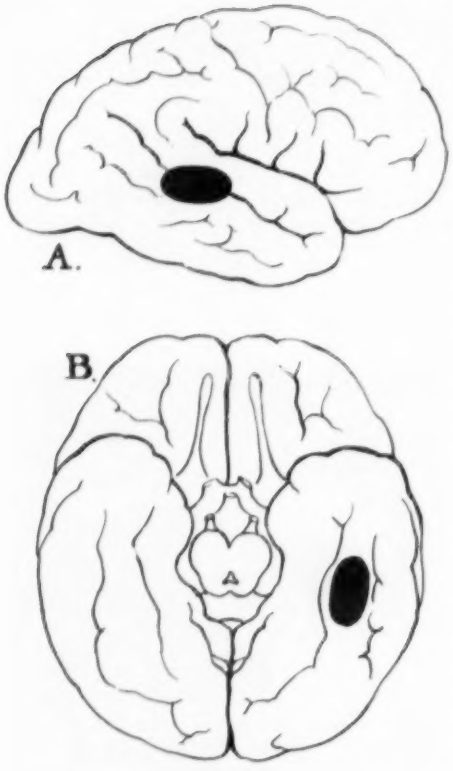


Fig. 32.—*A*, drawing of brain showing location of calcified tumor mass in the anterior one-third of the temporal lobe. *B*, horizontal section of tumor mass indicated in *A*.

terior portion of the right sphenoidal ridge (Fig. 18), which measured about 2.25 cm. in diameter, associated with a possible erosion of the right anterior clinoid process. The Stanford-Binet test revealed an I. Q. of 115. Ventriculography revealed dilated ventricles, with the right ventricle pulled to the right and enlarged, but not displaced. The electroencephalogram was normal. Eye examination revealed no abnormal findings except for a contraction of the right temporal field. Operation revealed that at the anterior end of the superior temporal gyrus, and under about 1 mm. of cortex, was a slick, well-encapsulated, yellowish, very firm tumor mass, which was completely removed. It was roughly spherical and about the size of an English walnut. It lay entirely in the anterior one-third of the temporal lobe, as shown in Figure *32A* and *B*.

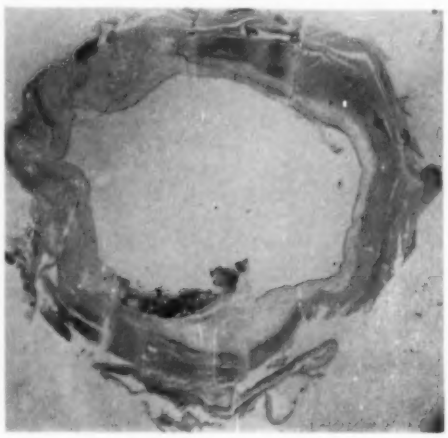
Pathologic examination revealed a circular, hollow mass of tissue containing many areas of calcification and consisting mainly of dense fibrous tissue with a very low nuclear content and a few scant vessels. No mitosis or amitosis was present, as is shown in Figure 33. A pathologic report of fibrillary astrocytoma (calcified) was made.

Case 8.—A 12-year-old white boy had no specific complaints and was seen in the clinic because of nervousness. The child was mentally retarded. There were no significant physical findings or laboratory studies except for the calcifications, as shown in Figures 19 and 20.

Case 9.—A 27-year-old white man gave the history of fronto-occipital headaches of nine weeks' duration, associated with what he termed a "fading of vision." Ten years previous to the time of admission he had consulted a doctor because of his small stature. The doctor had prescribed pituitary pills and thyroid extract. Physical examination revealed the patient's height to be 5 ft. 2 in., weight 140 lb., blood pressure 90/70. The basal metabolic rate was —22%, and on another occasion —27%.

There was a generous amount of fat quite evenly distributed over the thorax, trunk, and thighs. The forearms, hands, legs, and feet were small-boned and well muscled, with less subcutaneous fat. The skin was thin and relatively hairless. No facial or axillary hair was present, and there was very little hair around the pubis. The voice was high-pitched, and the genitalia were infantile in type. The skeleton was small-boned and of the childhood type. The head was tilted forward and the eyelids kept widely opened. The eyes seemed to stare at the floor, and the head was tilted slightly to the left. The olfactory nerve was intact, and the sense of smell was unimpaired. Eye findings revealed normal pupillary

Fig. 33.—Microscopic section of tumor, revealing a circular hollow mass of tissue containing many areas of calcification and consisting mainly of dense fibrous tissue with a very low nuclear content and a few scant vessels.



reactions, normal color perception, and paresis of left lateral gaze. Visual fields showed a superior temporal slant in the right eye and a constriction of the left temporal field. Skull x-rays showed a suprasellar calcification, as seen in Figure 21. A diagnosis of Fröhlich's syndrome, secondary to a suprasellar tumor, most likely a craniopharyngioma, was made, and a right transfrontal osteoplastic craniotomy was performed according to the Naffziger technique. At operation the scalp tissues were found to be very thin and soft, much like a child's. The bone of the skull was also very thin. After the dura was opened, the frontal lobe was carefully elevated and a large amount of fluid was encountered in the dilated basilar cisterns. The right optic nerve was very small and the chiasmal part brownish-blue in color. A cyst extended anterior to the chiasm, and upon opening it, a large amount of yellow fluid containing fatty deposits was emptied. Immediately below the chiasm and pressing upward into its structure was a large globular, irregular mass of cholesterol deposits, which extended into the chiasm. A good portion of the cyst wall, which extended downward into the floor of the skull in a large bony pocket, was removed. As much of the cholesterol crystals was removed as was possible without severing the chiasm. It was obvious that the chiasm rode on top of the calcified mass which was seen on the x-ray films. Four months postoperatively vision was 20/70 in each eye with bitemporal hemianopsia.

CASE 10.—A 64-year-old white woman, gave a history of turning in of the left eye of eight years' duration. Eight months prior to this hospital admission she had experienced the sudden onset of a buzzing noise in the left side of her head. General physical examination was noncontributory. The blood pressure was 140/80. Ocular findings revealed no exophthalmos or pulsations. The pupil of the left eye reacted sluggishly to light, and there was paresis of the left lateral rectus. Corneal sensation was normal on both sides. Fundi and visual fields were normal. Skull x-rays revealed an area of suprasellar calcification, as shown in Figure 22, in addition to erosion of the apex of the petrous portion of the left temporal bone and the left posterior clinoid process. Angiography was not performed. Ligation of the left common carotid artery was performed, and the patient's postoperative course was uneventful. However, as late as two years after operation some paresis of the left lateral rectus persisted.

Cases 1, 7, 9, and 10 and Figures 8, 17, 18, 21, and 22 are published with the kind permission of Dr. Loyal Davis, Department of Neurosurgery, Northwestern University Medical School.

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A New Ptosis Operation Utilizing Both Levator and Frontalis

ROBERT A. SCHIMEK, M.D., Detroit

The 80-odd varieties[1] of ptosis operations are usually divided into four groups. These groups are classically based on (1) enhanced use of the levator muscle, (2) use of the frontalis, (3) use of the superior rectus, and (4) skin excision methods. The principles of these methods, with their advantages, disadvantages, and modifications, have been adequately described in the literature. Skin excision methods may be dismissed as neither permanent nor practical, except in cases of blepharochalasis. Use of the superior rectus risks exposure keratitis from interference with normal blinking and incomplete lid closure during sleep. Other complications which may occur include diplopia and hypotropia, arching of the lid margin, entropion and trichiasis, and excessive postoperative scarring. Use of the levator and use of the frontalis are deservedly the most popular methods.

Frontalis motion can be transmitted to the tarsal border of the lid by permanent sutures, temporary sutures forming scar tracts, flaps of skin or fascia lata, and muscle strips of orbicularis or of frontalis. This type of operation gives the lid voluntary movement in the absence of levator function. Disadvantages are the peculiar facial expression which may result from use of the frontalis, the lack of complete synchronization of movement between the lid and the eye when the eye is rotated vertically, danger of exposure keratitis during the early postoperative period, and the appearance of vertical lid folds with a deeply set eye.

Operations which enhance levator action, as in levator resection, have the great advantage of permitting almost normal blinking and synchronous following movements of the lid with the eye. There is a relatively small incidence of excessive postoperative scarring, lagophthalmos, deformation of the lid margins, diplopia induced by operation, or unsightly asymmetrical elevation of the brow. In general, with levator action present, levator resection gives the most ideal results. However, if there is no levator action, opinion varies as to the efficacy of levator resection. Blaskovics[2] stated that "even when the muscle is absent the operation can be performed and be effective . . . an apparent function develops even in those cases in which this muscle is completely paralyzed or atrophic." On the other hand, most authors agree that levator function must be present preoperatively; otherwise, levator resection will give inadequate correction or, at best, correction with poor lid mobility. Berke and Wadsworth[3] have shown that in cases of congenital ptosis with poor levator action the levator frequently lacks striated muscle fibers and levator resection often gives a poor result.

In cases with poor levator function preoperatively, it should be advantageous to combine actual use of the levator with a frontalis type of operation. One would like to enhance levator function to the maximum degree and still obtain accessory frontalis action. This accessory frontalis action can compensate for any undercorrection that might result from doing a simple levator resection. A few operative techniques can be found in the literature which combine the use of the levator and

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From the Department of Ophthalmology of the Henry Ford Hospital.

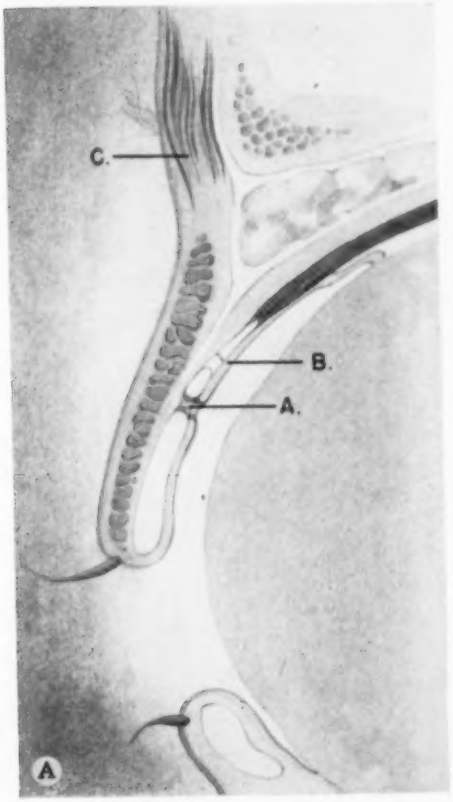

Fig. 1.—*A*, cross section of upper lid, showing structures in normal relationship, but with preliminary incisions made (*A*). Buttonhole in tarsus through which levator tongue will pass; (*B*) incised edge of levator tendon at its insertion to the upper tarsal plate. (*C*) frontalis muscle. The tendinous portion of the levator is much longer than shown.

frontalis,* but these have not been generally adopted.

During the past two years, a new operation for severe ptosis with poor levator action has been under evaluation.[5] This operation combines the advantages of (1) enhanced use of the levator muscle, (2) utilization of the frontalis muscle, and (3) use of the connective tissue framework of the levator. The last is particularly important if the levator is markedly paretic. For ptosis with poor levator action, combining these three methods is advantageous and is accomplished in the following manner: The levator is incised from its tarsal insertion, dissected from the lid, and split longitudinally (Fig. 2*A-G*). Two horizontal buttonholes are incised through the tarsus (Fig. 2*H*). Two skin incisions are made above the brow (Fig. 2*I*). A ptosis knife, passed subcutaneously from each brow incision to the corresponding tarsal buttonhole, is used to pull each band of levator through the tarsal buttonhole up to the frontalis muscle, to which

B, cross section of lid, showing altered relationships resulting from the operation. The levator muscle has been advanced through the tarsal buttonholes and passed subcutaneously up to the frontalis muscle, to which it is sutured. This elevates the lid, owing to (1) effective advancement of the levator muscle, (2) direct transmission of frontalis action to the lid, and (3) use of the connective tissue matrix of the levator. The levator structure which extends from each tarsal buttonhole to the frontalis is mostly tendon.

* References 4 and 5. Angelucci, A.: Paper read at 13th International Congress of Medicine, Paris, 1900; cited by Beard.[5]

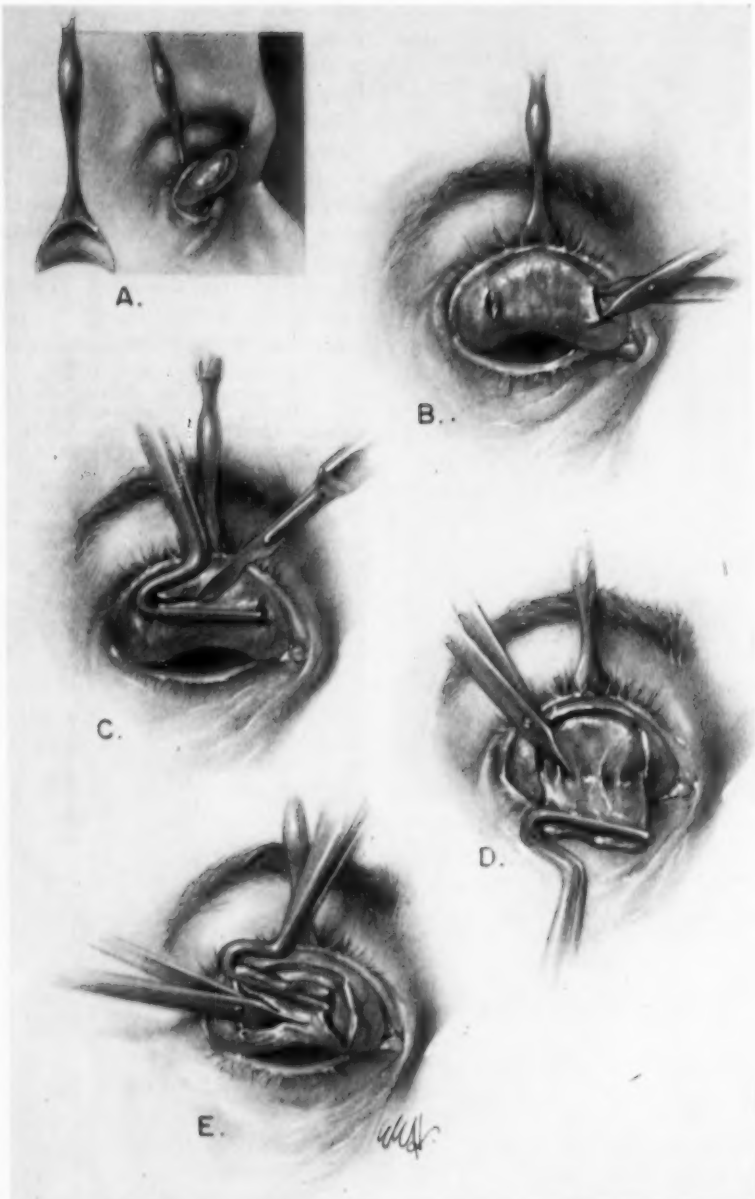

Fig. 2.—Operative technique: (*A*) The lid is everted. (*B*) Temporal and nasal buttonholes are cut through conjunctiva and levator above the tarsus. (*C, D*) Conjunctiva, Müller's muscle, and levator are clamped and separated as one layer from the lid structures. (*E*) Conjunctiva is peeled from the distal levator tendon to be buried in the lid.

it is sutured (Fig. 2*J*). As a result of this technique, the two bands of levator pass as a sling through the tarsus to the frontalis (Fig. 1*A, B*).

This technique increases levator power by advancing or effectively shortening the levator muscle fibers, thus accomplishing the most physiologically possible correction of

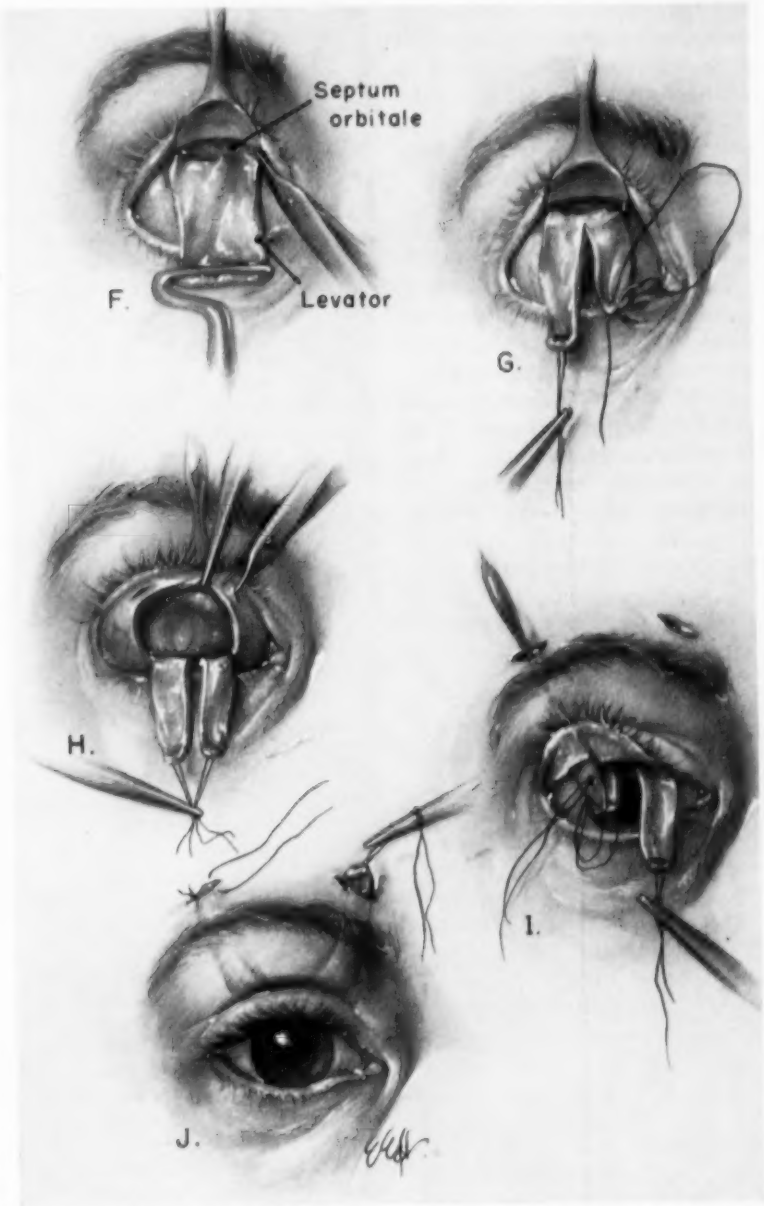

Fig. 2 (cont.).—(*F*) The medial and lateral horns of the levator are cut. (*G*) The levator tendon is split longitudinally and a suture passed through each tongue. (*H*) Two small buttonholes are cut in the tarsus. (*I*) A ptosis knife is passed subcutaneously from each of the two brow incisions out through the corresponding tarsal plate buttonhole. (*J*) By withdrawing the ptosis knife (with the levator suture through the hole in the ptosis knife), each tongue of levator is pulled up to the frontalis muscles, to which it is sutured. The skin incisions are sutured.

the ptosis. It preserves almost normal blinking, lid closure, and synchronous following movements of the lid with the eye.

Supplemental use of the frontalis compensates for any undercorrection that might result from utilization of a paretic levator

alone. The frontalis-tarsus connection by levator tendon, rather than a buried suture or a fascia transplant, avoids the risk of infection around foreign material at a later date.

The operation also utilizes the connective tissue matrix existing in a paretic levator muscle for elevating the lid. This factor is important when there is very poor levator function and the muscle content of the levator is greatly reduced. When a Blaskovics type of operation is performed on a paretic levator muscle, either a marked degree of lagophthalmos (from use of the connective tissue matrix of the paretic levator muscle to suspend the lid) or, more frequently, an inadequate correction results. The poorer the levator action, the more inelastic and rigid is the levator tissue matrix, and the more marked the tendency to inadequate correction and lagophthalmos. In this operation, the connective tissue support of a paretic levator is used, but the addition of voluntary frontalis movement permits increased elevation and mobility of the lid.

TECHNIQUE

The operative technique has two phases. The first phase consists of dissecting the levator from the other lid structures. This is done by a modified technique similar to the one used for many years by Guyton † and by Verhoeff ‡ and recently recommended by Iliff.[7] This modified technique is quick and simple with very little bleeding, as compared with the original Blaskovics method. However, the original Blaskovics method can be substituted if one desires. The second phase of the operation consists of advancing the levator in sling-like fashion through buttonholes in the tarsus and attaching it to the frontalis. This anatomical change is shown in cross section in Figure 1*A* and *B*.

DETAILED DESCRIPTION OF TECHNIQUE

General anesthesia may be used routinely, or local injection may be substituted in adults. Each step of the technique is lettered to correspond with the operative illustrations in Figure 2.

(*A*) The upper lid is everted with a lid holder.

(*B*) With sharp-pointed scissors, a temporal buttonhole and a nasal buttonhole are cut through conjunctiva and levator above the tarsus.

(*C*) One jaw of a Berke ptosis clamp is then inserted into the temporal buttonhole, then passed across the lid beneath conjunctiva and levator, and out through the nasal buttonhole. The jaws are clamped together. An incision is made between the clamp and the border of the tarsus.

(*D*) By sharp and blunt dissection, the levator, Müller's muscle, and the conjunctiva are separated as one layer from the septum orbitale and lid structures to the level of the upper fornix. Bleeding is minimal because conjunctiva, Müller's muscle, and levator (1) are clamped close to their cut borders and (2) are easily separated from the septum orbitale by blunt dissection.

(*E*) With blunt dissection by scissors, conjunctiva is easily peeled from the portion of the levator tendon to be buried in the lid. In the illustration, the upper clamp holds conjunctiva, Müller's muscle, and levator. The scissors are cutting conjunctiva, which will be peeled off from the distal 1.6 cm. of the levator tendon and discarded.

(*F*) The medial and lateral horns of the levator are cut. This is facilitated by (1) reinserting the lid retractor as a rake to retract the upper lid and (2) stretching the levator downward.

(*G*) The levator is split longitudinally for about 1.6 cm. A double-armed mattress suture of 4-0 black silk is passed through each tongue of levator.

(*H*) The lid is again everted on a lid holder. Two small buttonholes are cut in the tarsus 2 mm. below and parallel to the superior tarsal border at the center of the inner half and the center of the outer half of the tarsus respectively.

(*I*) Two horizontal skin incisions are made, respectively, medially and laterally 0.5 cm. above the brow. Blunt-pointed scissors are inserted deeply through the incisions toward the tarsus and spread to begin a passage from the brow incision to the tarsal buttonholes. A ptosis knife is inserted through each brow incision, is passed deeply over the frontalis muscle, and is then passed superficially (just under the skin) in the lid to a point 3 mm. above the lash line. The ptosis knife is then directed out through the corresponding tarsal plate buttonhole. It is important to keep as close as possible to the skin of the lid to obtain a good position of the lashes and a good lid fold. The sutures from each tongue of levator are passed through the hole in the blade of the ptosis knife.

(*J*) By withdrawing the ptosis knife, each tongue of levator is pulled through its tarsal buttonhole up

† Guyton, J. S.: Personal communication to the author.

‡ Verhoeff, F. H.: Personal communication to the author.

Fig. 3.—*A*, bilateral congenital ptosis with marked undercorrection and lid scarring following Hunt-Tansley operation. Postoperatively, ptosis was satisfactorily corrected in the primary position (*B*). There was only slight lid lag in downward gaze, and lid closure was satisfactory.

to the frontalis muscle and the corresponding brow incision. Each levator tongue is sutured to the frontalis muscle. This is facilitated by lifting a deep bundle of frontalis muscle and fascia into the brow incision with a skin hook. A curved needle threaded on one arm of the levator mattress suture is passed deeply through this bundle of frontalis and tied. For a marked ptosis with poor levator function, the suture is tightly tied, lifting the lid border. The suture ends are cut and allowed to retract and remain buried. The brow incisions are closed with 6-0 black silk sutures. The lower lid should be pulled up by Frost sutures to protect the cornea.

POSTOPERATIVE MANAGEMENT

The patient may be discharged from the hospital on the first day after operation. The Frost sutures may be removed at discharge or allowed to remain several days to assure protection of the cornea during the initial postoperative period. The skin sutures closing the brow incision are removed on the fourth or fifth postoperative day. In children, the eye should be covered with a tight adhesive dressing until the brow skin sutures are removed. In the initial postoperative period, a certain amount of lagophthalmos is the rule if a complete correction of the ptosis is to be achieved. This is especially true in cases with very poor levator function. This initial lagophthalmos diminishes.

Usually there is very little postoperative swelling and reaction. If this is the case,

Fig. 4.—Left congenital ptosis without levator action, with history of two previous unsuccessful operations (tarsal resection by the Eversbusch technique and a Friedenwald-Guyton suture). Postoperatively, an excellent result was obtained in the primary position (*B*). There was only slight lag of the surgically treated lid on upward gaze (*A*), and lid closure was satisfactory (*C*).

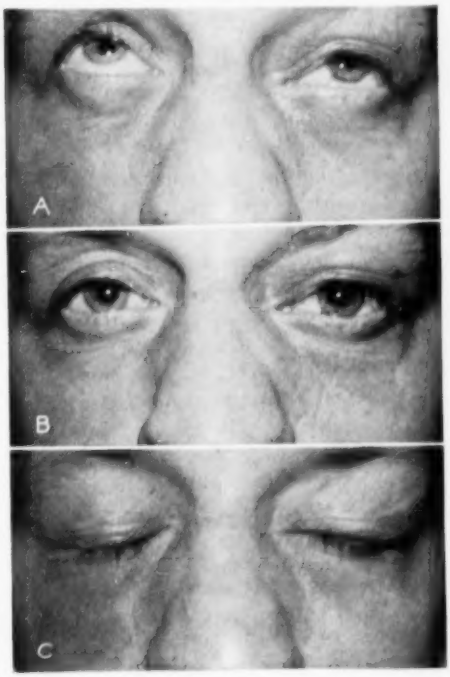

Fig. 5.—Marked congenital ptosis of the right eye. Preoperatively (*A*), there was no apparent levator action on looking up. The postoperative result was excellent (*B*) with only minimal lid lag on looking up and moderate lid lag on looking down, and with satisfactory lid closure (three weeks after operation). The slight lid lag on downward gaze diminished postoperatively.

a slight overcorrection of 1 or 2 mm. is desirable during the first few postoperative weeks. If there is a marked amount of ecchymosis or swelling, there may be an initial undercorrection, due to secondary swelling of the lid tissues. In the latter case, the effect of the ptosis operation will increase during the next month, just as the effect of a Blaskovics operation will often increase postoperatively.

REVIEW OF CASES

Six ptosis operations utilizing this method have been performed in a total of five patients. All these patients had Grade 4 ptosis with poor or no levator function preoperatively. Satisfactory results were obtained in all cases. With such cases, perfectly normal levator action was not and will not usually be achieved. However, the maximum levator action that it is possible to gain in such cases will be attained with auxiliary frontalis action. The following cases all showed increased levator action postoperatively.

Case 1.—A 15-year-old white boy with congenital bilateral ptosis had had a bilateral Hunt-Tansley operation. When seen nine years later, he had marked residual ptosis without apparent levator action (Fig. 3). The operation described in this report was performed on both eyes, with a good result, as shown in Figure 3. There have been no complications during the nine-month follow-up observation period.

Case 2.—A 45-year-old white man had two previous unsuccessful operations for congenital ptosis of the left eye. A resection of the tarsus by the Eversbusch technique had failed, and a Friedenwald-Guyton suture also failed when the tantalum wire broke. Preoperatively, the patient had a marked Grade 4 ptosis with no apparent levator action. Postoperatively, an excellent result was obtained, as shown in Figure 4. There have been no complications during a follow-up period of one and one-half years.

Case 3.—A 6-year-old white boy had Grade 4 congenital ptosis of the right upper lid with no levator action preoperatively (Fig. 5*A*). An excellent postoperative result was obtained (Fig. 5*B*). The operation was performed by another surgeon,

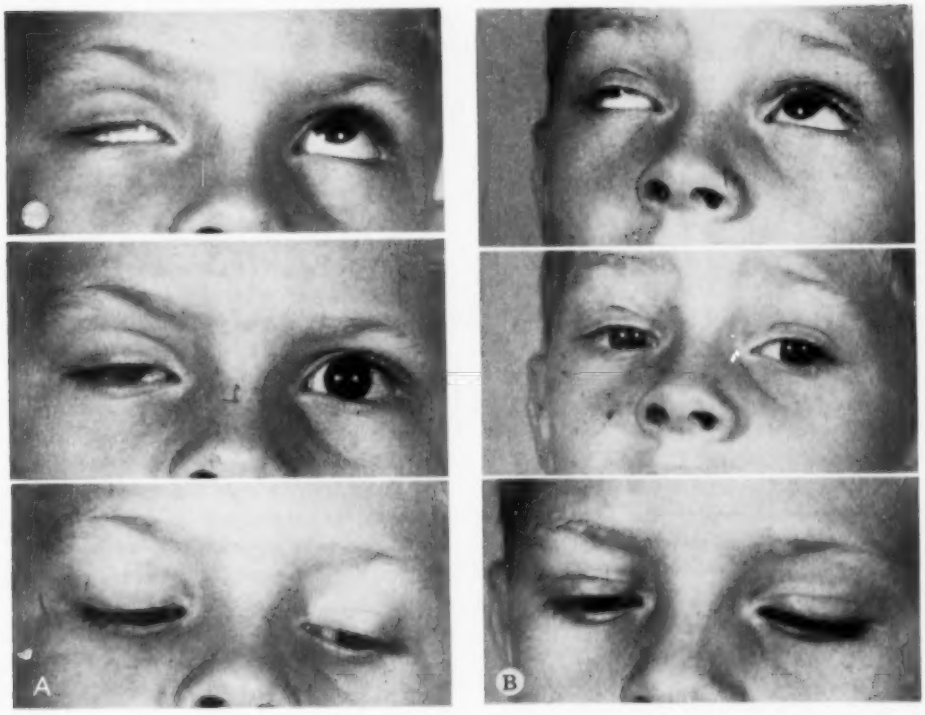

Fig. 6.—Marked congenital ptosis on right (*A*), with satisfactory postoperative result in primary position and on downward gaze (*B*). Moderate lid lag on upward gaze.

and the conjunctival epithelium was not removed. About six months later two epithelium-lined cysts which occurred beneath the skin of the lids had to be excised. Otherwise, there were no complications, and the final result was satisfactory. This case emphasizes the necessity for removing conjunctival epithelium from the portion of the levator to be buried in the lid, as was done in all the other cases.

CASE 4.—A 5-year-old white boy had a marked congenital ptosis on the right without apparent levator function (Fig. 6). The skin incisions were made in the brow instead of above the brow, resulting in an insufficient advancement of the levator and marked lid lag on upward gaze. Otherwise, a satisfactory postoperative result was achieved, with no complication occurring during the nine months following operation.

SUMMARY

A new ptosis operation which utilizes both levator and frontalis is described. The operation has been successfully used for Grade 4 ptosis with very poor or no levator action.

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Annual Reviews

STRABISMUS

Review of the Literature

MAYNARD C. WHEELER, M.D., New York

ONCE AGAIN I have tried to review all of the significant articles on strabismus and related subjects that have appeared during the preceding year. Undoubtedly I have missed some important ones. Some foreign articles have not been available in the original, or I have been unable to obtain satisfactory translations. After writing this review for three years, I am convinced that it cannot be written as a continuous story, as I had originally hoped to do. While the bulk of material is certainly sufficient, the truly significant advances in any one year are too few to enable one even to approximate a coverage which could be termed "recent advances."

BINOCULAR VISION

Fusion.—The fusion of 35 doctors and students with normal vision was studied by Berger,[1] using Bielschowsky's prism apparatus. Vertical and torsional phorias were shown by 30%. She believes that fusion of vertical double images is obtained by muscular cooperation, while fusion of torsional double images takes place by sensory coordination.

Peripheral Fusion. — Winkelman[2] has briefly discussed his work on peripheral and macular fusion for the British orthoptists. The position and size of the peripheral object provide a means of measuring the strength of the fusion of the maculas. Peripheral fusion offers a means of understanding more fully the fusion mechanism; so it may be of great importance in orthoptic practice. He believes that peripheral fusion probably holds the eyes of babies straight, as well as the eyes of patients whose central vision is lost in one eye (Dobson[72]).

Anomalous Correspondence.—Discussions of various phases of anomalous correspondence again occupy a prominent position in the literature, although most of the articles include so much material on the treatment by orthoptics and the results of treatment that they are reviewed later under "orthoptics." The majority of authors accept anomalous correspondence as an adaptive mechanism, but there is still a wide difference of opinion as to the incidence, due undoubtedly to the kinds and interpretations of the tests used and to the absence of a generally accepted definition.

An amazingly low incidence is reported by François and James[3]—1% of 1300 strabismus cases. Of the patients, 37.8% had no binocular vision, probably an absence of functional correspondence due to profound suppression. Emphasizing the distinction between suppression, which is essentially monocular, and anomalous correspondence, which is essentially binocular, they state that suppression may be partial or total, the latter having either loss of central vision (5.3%) or eccentric fixation (1.46%). In cases of loss of central vision they consider the prognosis excellent up to the age of 8 or 10 years, using constant occlusion and orthoptics, while eccentric fixation is incurable. They draw a sharp distinction between loss of normal correspondence, true anomalous correspondence, and absence of binocular vision. Loss of normal correspondence is often curable by appropriate exercises, while true anomalous correspondence is never curable. They consider that the reports of cures of anomalous correspondence are really the cases that they classify as having lost normal correspondence. After operation in cases with anomalous correspondence, a false binocular vision may be acquired that is sufficient to

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prevent a reappearance of the strabismus. Their concept of true anomalous correspondence is orthodox, but in spite of considerable discussion their definition of cases which they classify as those of loss of normal correspondence is not clear. The much larger group (according to their figures) of absence of binocular vision is not described. These authors appear to have an excellent understanding of the anomalies of binocular vision in strabismus. Their distinction between true anomalous correspondence, loss of normal correspondence, and loss of binocular vision may have significance. It is hoped that they will tell us more about it.

Levinge * has found the results of the treatment of anomalous correspondence unsuccessful, in general. Either the condition is unchanged or the degree of binocular vision is too weak for a functional result. She has tried to devise a test for anomalous correspondence that reproduces ordinary visual conditions, believing that it will be more likely to elicit a response showing the usual adaptation of the patient. She calls it the "projection test." A patient wearing polarized goggles fixes an oblong light, 2×4 in. (5×10 cm.) at 1 meter. Half of the oblong, plus a control mark, is seen by each eye. There are four possible responses with manifest strabismus: 1. The light is seen as two sections with the separation corresponding to the angle of strabismus: normal correspondence. 2. The light is seen as two sections, but the separation does not correspond to the angle of strabismus: unharmonious anomalous correspondence. 3. The light is seen complete: harmonious anomalous correspondence. 4. Only one section of the light is seen: complete suppression, unilateral or alternating. Levinge also uses the following tests: red-glass, after-image, synoptophore, subjective cover, fusional response with vertical prism, physiological diplopia, and binocular convergence. She finds the synoptophore inadequate as the sole test for correspondence in small deviations and where there are large areas of suppression. Her projection test gave more harmonious anomalous correspondence. The red-glass test seemed most satisfactory. Her conclusions are very interesting. She believes that anomalous correspondence is undoubtedly binocular, bringing advantages of improved binocular response and wider visual fields. It is a physiological part of normal binocular vision in many persons and is a natural accompaniment of binocular adjustment when the visual axes are not parallel. Anomalous correspondence should be harmonious. The failure to find this in every case may be due to the inadequacies of present tests. For its development there must be a constant deviation, intelligence, and adaptability, and probably some binocular vision must have been present prior to the onset. Orthoptic treatment is not only unsuccessful but ill-advised, since it may deprive the patient of the advantage of a binocular condition. The author has been treating patients by using their abnormal correspondence in an attempt to improve binocular function. It has been beneficial in adults with poor binocular convergence who have symptoms. Her basic treatment is to increase the range of fusion, using abnormally corresponding points. (For another who shares this view, at least partially, see Hamburger.)

Pointing out that the problem of fusion is the problem of strabismus, de Jaeger and Bernolet * believe that the understanding of the anomalies of fusion enables them to make a prognosis in and treat strabismus. If strabismus comes on very early, before fusion has developed, or late, after it is fully developed and extinction is no longer possible, it will be alternating in type. Suppression is a conditioned reflex for alternating strabismus, while it has become unconditioned or obligatory for monocular strabismus. Suppression is stronger the smaller the angle and the better the visual acuity. The patient adapts to the deviation so that he can project correctly. The more harmonious and fixed the correspondence, the more difficult it is to cure. Anomalous correspondence is less well established in monocular strabismus with amblyopia. The authors can measure the depth

* References 4 and 5.

of adaptation of the strabismus patient to his state and thus make a prognosis.

A new method for testing retinal correspondence is described by Falkowska.[7] A 60 cm. board is placed in the central vertical plane in front of the patient's face. A straight line is drawn on each side of the board, at the same level, so that one line is seen with each eye. Openings are cut in the board at different distances so that a fixation object placed in the opening can be seen by both eyes. A detailed description is given of what happens to the relative positions of the lines and the fixation objects in different conditions of correspondence.

The "after-image transfer test," described last year by Jaffe, has been investigated by Hansen,[8] using only patients who had anomalous correspondence with the after-image test. Only 3 of 18 patients failed to transfer the after-image to the unexposed eye. He states that after-images are a function of the retina and occipital cortical centers. When the subject is aware of the after-image, when the unexposed eye is open and the exposed eye closed, it does not mean that the unexposed eye has begun to send impulses to the cortex or that the eye is aware of the after-image. It means only that the exposed eye continues to produce a visual sensation, regardless of whether the eyes are open or closed. This is proved, because the after-image is always positive when the exposed eye is closed and the unexposed eye open. Persons with anomalous correspondence do not respond differently in after-image transfer than those with normal correspondence. Hansen explains those not aware of the after-image in the unexposed eye by strong suppression and by visual impressions arising in the open, unexposed eye which overwhelm the impulses coming from the exposed closed eye. This explanation sounds logical and confirms our experience with this test.

AMBLYOPIA

The resolving power of the fovea and the accuracy of fixation in amblyopic eyes have been investigated by Pugh,[9] hoping to find further evidence to localize the site of the inhibition leading to the reduced visual acuity in amblyopia. In 100 cases studied in detail, the vision in the amblyopic eyes ranged from perception of hand movements to 6/12, and the ages, from 10 to 55. The orthoptoscope was used with an arrangement that permitted reducing the light before the good eye to 1/10,000. With use of Chavasse's retinal rivalry slides, which vary from 5 degrees to 5 minutes, the amblyopic eye was tested first; then when binocular tests were made, the acuity of the amblyopic eye dropped markedly. With monocular tests, there was an alteration in acuity with different directions of regard in many cases—some with the eye adducted, some with the eye abducted, but always in the same direction, an anomaly which Pugh called "directional preference." She believes that the cortex has been demonstrated to be the probable site of inhibition in about 50% of this number; 76% had 5 degrees of deviation or less and all had normal correspondence, but 12% showed directional preference. Of the other 50%, cortical inhibition was demonstrated in 52% with definite anomalous correspondence and in 92% with indefinite retinal correspondence. Of the cases of marked anomalous correspondence, 76% had a deviation of over 5 degrees, and all had directional anomalies. The linkage between the angle of deviation, retinal correspondence, and cortical suppression suggests the probability of some local change in the photoreceptors of the fovea, and thus an important site of defective foveal vision. Pugh found the correlation between the amount of deviation and the anomalies of foveal response difficult to explain. She believes that the possibility of local changes in the fovea being due to continued deviation is worth further investigation.

An ingenious way of testing the vision of very young children has been devised by Schwarting.[10] It is based on the facts that in darkness a baby will direct his gaze toward the sudden appearance of an illuminated field, provided it is not bright and glaring, and that a visible object moving across the field will usually elicit the following reflex of the eyes. In the author's device,

steel wires of various sizes are moved by a metronome in front of an illuminated arc, at a constant speed of 40 half-cycles per minute. Synchronous movement of the eyes can be observed if the child sees the wire. Schwarting has correlated the test with the Snellen chart by testing many adults. He has frequently been able to check the vision of babies monocularly.

A new scale of test figures for small children is presented by Rossano,[11] but, unfortunately, the article is not illustrated. The test is an album of silhouettes familiar to children. His purpose is not to measure visual acuity but to compare the eyes, in order to detect amblyopia.

Treatment.—Last year, when his monograph on this subject appeared, Bangerter's name was virtually unknown in this country. This year there has been hardly an article dealing with the treatment of amblyopia that did not at least mention his pleoptic treatment. It is not surprising that his method of treating eccentric fixation has been readily accepted, because no other treatment has been known. His idea of a school for training these patients has also been widely accepted in principle, although most authors, while convinced that the superior results achieved justify the much greater expense, have been unable to finance similar projects (Rintelen and Hotz,[66] Meitinger,[67] Hamburger[65]). Possibly it can be said that a modest beginning has been made in this direction in that several amblyopia clinics have been established. It is claimed that these clinics reduce the time required for occlusion. James[12] has the children attend her clinic twice a week in groups of five or six. York[13] describes such a clinic, the only one of the sort in Sydney. As part of an orthoptic department, amblyopic patients are seen one afternoon a week, coming weekly for two visits, then monthly. If there is no improvement for three months, the child is sent back to the doctor. She considers the amblyopes the greatest reason for an orthoptic department and their cure the greatest achievement.

In a new paper, Bangerter[14] goes briefly over the material of his monograph. He emphasizes again that occlusion is used only when a satisfactory central faculty of vision has been achieved by other means; otherwise, occlusion usually will promote the development of paracentral fixation with inferior visual acuity. Secondary amblyopia is harmless, since once the faculty of vision has existed it may become dormant but will never be lost. The most important point in early pleoptic treatment is creation of a reserve.

A modification of Bangerter's interesting method for correction of false macula in strabismus by producing a scotoma at the false macula, by strong illumination, then stimulating the true macula, while the scotoma persists, is described by Castanera and Muinos.[15] Instead of occluding the good eye and depending on the false macula for fixation, they use a major amblyoscope to control the fixation of the good eye. They are able to stimulate the true macula of the squinting eye for 10 to 20 minutes, 6 to 12 times a day. Twelve of 21 patients recovered normal fixation in a short time; in the other 9 the false macula was absolute.

Except for the cases of eccentric (paramacular) fixation, constant occlusion is the approved method of treating amblyopia by almost all authors. One exception to this is the experience of Nemetz, Pressina, and Schmidt † at the first University Eye Clinic in Vienna. They emphasize the difficulties that we have all experienced with constant occlusion, but they have been more discouraged than most. They seldom achieved visual acuity of better than 6/12 by occlusion. Inspired by Bangerter's work, they had his "amblyopia apparaus" made in Vienna. In a small series of cases they report better results with this apparatus, after occlusion had been tried; this improvement was more striking in the cases with paramacular fixation. Because of the difficulty of getting patients to come regularly for work on the apparatus, they advocate an occluder made of adhesive tape applied to both sides of the spectacle

† References 16 and 17.

lens and extending well beyond it, but trimmed accurately to fit the face. They do not tell us how they keep the glasses securely in position.

After four years in the orthoptic department of the University Eye Clinic in Zurich, Braun and Boissonnas [18] report some of their impressions. Claiming that Bangerter can put more emphasis on monocular training because he has more help, they still continue total occlusion, during which the children attend the clinic two to four times a week for intensive training. Occasionally a child is given monocular training as an inpatient. If the parents cooperate, their results are as good as those obtained at St. Gall (Bangerter). The authors close by saying that "we could never work on monocular treatment to the same extent as St. Gall."

Rønne [19] also insists on complete occlusion. In children too young for vision testing, he continues occlusion until they show alternation. He stresses the importance of prophylaxis and early treatment, believing that amblyopia can be prevented. He gives his results in 270 cases, in which vision of 6/9 or better is considered a success.

No. of Cases	Vision	Fixation	Success
61	Less than 3/60	Uncertain or eccentric	61%
64	6/60 or less	Central	73%
100	6/36 to 6/18	Central	93%
50	Too young for original vision but undoubtedly amblyopic		100%

These figures are most impressive and should go a long way toward convincing any who are still skeptical about the treatment of amblyopia. They would be even more valuable if Rønne had given the comparative vision of the two eyes at the end of treatment.

Hammer [20] has treated 1040 patients with strabismus since the war, of whom 44.8% were amblyopic. He estimates that about 1.5% of the total population are amblyopic and states that there is a greater tendency to accidents among amblyopes. (Nemetz, Pressina, and Schmidt concur in both these statements.) Systematic training was given to 285 patients with monocular strabismus with amblyopia. Of these, 89.9% were improved, 7.7% were unchanged, and 2.5% became worse. Hammer starts the treatment with occlusion for three months, followed by atropinization and prescription of glasses, as well as the use of special practice glasses with Wickert's strabismus capsule (not described). He has devised a new "calotte" (also not described) that will make the practice glasses unnecessary as soon as it is available. He stresses systematic daily practice, which should be continued during the first year of school. He has enlisted the teachers to supervise the work with the practice glasses. The importance of educating the parents, particularly because of the neglect of amblyopia after operation, is emphasized. He trains children in groups, to overcome their reluctance to wear occluders.

Believing that in the majority of cases strabismus appears before one year of age, de Jaeger and Bernolet [21] find that in 67% of cases the strabismus is unilateral with amblyopia and that there is no tendency toward improvement without treatment. They favor operation before the age of 3 years in order to allow the eye to develop macular function. They use constant occlusion with exercises after 3 years, and get cures in 90% of amblyopic patients if treated between 3 and 4 years of age. After 5 years of age only 60% are cured.

OCULAR MOTILITY

An important study of primary and auxiliary medial ocular rotation has been reported by Gordon.[22] He has demonstrated that the arc of contact of the medial rectus will permit, by its unwrapping mechanism, approximately 35 degrees of internal rotation. This was measured on cadavers. Internal rotation beyond this must be provided by another mechanism, i. e., the superior and inferior recti. He cut the superior and inferior recti on six patients to demonstrate this. I believe that this is the first time that this mechanism has actually been verified on patients. Incidentally, he found that when these vertical recti were separated from the globe, they become rapidly reattached, in spite of large myectomies. He suggests that

this will explain some of the unexpected results from muscle surgery.

Torsion.—Quereau [23] admits that the objective detection of torsion is difficult. With the other eye covered, he picks a conjunctival vessel that is on the line from the center of the pupil along which the eye is going to rotate. The observer sights along this line. Most of the torsion seen objectively is caused by action of the synergist of the paretic muscle. The plane of action identified by corneal torsion is the plane of action of the synergist. This method should be used as an adjunct to the cover test, together with Bielschowsky's head-tilting test.

True torsion is described by Pascal [24] as a wheel-like rotation of the eye during which corresponding corneal and retinal meridians rotate in the same direction. False torsion occurs as a component of rotation of the eye on an oblique axis, the corneal and retinal meridians being inclined in different directions. Raubitschek [25] agrees with Pascal, but Roelofs [26] disagrees. Apparently, this disagreement comes from using planar coordinates. Quereau finds the corneal and retinal meridians tilt in the same direction when spherical coordinates are used.

EXAMINATION AND DIAGNOSIS ‡

Hugonnier [27] has given a detailed, and generally excellent, outline for the examination of children with ocular deviations. At the beginning he points out that if one is satisfied with a cosmetic cure the examination can be sketchy, while if one is hoping for a restoration of binocular vision it must be complete and detailed. His outline cannot be abstracted, but a few features deserve comment. It is gratifying to see that he emphasizes the history. He is dissatisfied with the "E" test; he judges the vision by the fixation in the young and stresses the comparison of the two eyes. He considers the prism cover test fundamental. The Krimsky prism reflex test is used for amblyopes. He leans heavily on the major amblyoscope for measurement and study of binocular vision.

‡ See Torsion.

The Hess and Lancaster methods are still being compared, combined, and modified. Foster, Pemberton, and Bagshaw [28] believe that the Lancaster method is superior because of its greater sensitivity, its ability to show cyclophoria, and its lower liability to error in cases of lack of normal correspondence. They have modified it by using sheet iron painted gray with 11- and 28-in. white squares painted on it. White, red, and green torches and three sets of colored magnets are used. The test is done in semidarkness at 0.5 meter. First, diplopia is looked for with the white light. Then, with the patient wearing the red-green goggles, with the red glass over the right eye, the test is conducted in the usual way. It is finally repeated with the torches reversed. The authors claim that the advantages are in time, cost, and interpretation. A single chart contains three diagrams, giving a much clearer picture than two Hess charts and a diplopia record. In his modification, Barthelmess [29] uses two projectors. The patient projects a green arrow, while the Hess figure is projected in red. He finds that it is easier to get and to keep complementary colors with the projectors. The images can be equalized with filters, and it is possible to work at a greater distance, thus making the method more delicate.

The method of concordances of Marquez has been enlarged and adapted by Pascal § for the diagnosis of strabismus. Using red-green spectacles, a small round light, and a linear light, he performs five diplopia tests. 1. The round light before the fixing eye—which eye is higher? 2. The round light in the four diagnostic corners—where is the greatest vertical separation? 3. The light straight ahead; head tilted toward the right and left—where does vertical separation increase? 4. The light on the side of least separation in Test 2—horizontal relation of two images? 5. The vertical line as in Test 4—which image is tilted and how? He then utilizes his "Benzene Muscle Ring Schema" to interpret these findings. He believes that it is best for the diagnosis of paralysis or

§ References 30 and 31.

spasm of a vertical muscle, but that it is useful for horizontal muscles. The secondary actions of the horizontal recti are easily visualized.

Electromyography.—The electrical recording of the eye movements, the electromyogram (EMG) or the electro-oculogram (EOG), is unquestionably attracting increasing interest, as seven papers on this subject have appeared in the literature. Björk's [32] interest is chiefly in neurogenic pareses. He believes that the EMG supplies information of great importance in the understanding of the physiology of the muscular system. He has a routine clinical method that is widely used in diagnosis. He investigated 24 patients with pareses due to lesions of the peripheral motor neurone and summarizes his results as follows: 1. The number of action potentials were reduced; the duration and form were like those of nonparetic cases. 2. Fibrillations were difficult to diagnose. 3. Uneven activation of residual action potentials was of characteristic appearance in a number of cases. 4. One could tell whether the paralysis was total by the EMG, and the course of a severe paresis could be followed. In moderately pronounced or mild cases, no reduction in the number of action potentials was shown. 5. It was shown that the EMG assists in differentiating between ocular muscle paresis and mechanical factors. It is an aid in the diagnosis of congenital limitation of movement.

In a second paper Björk [33] found that the lateral and medial recti show pronounced activity on forward gaze, and even in extremes of gaze the activity in the antagonist does not disappear entirely. In abducens palsy the electrical activity in the antagonist medial rectus was normal. Activity decreased in the direction of the paretic eye and almost disappeared when the nonparetic eye turned strongly in this direction, even though the paretic eye had not reached the midline. Compulsory movement of an eye with normal motility (passive movement with a bridle suture) showed no distinct change of activity.

A method for taking EMG's with a neuromuscular unit is described by Kuboki.[34]

Magee [35] enumerates several possible types of electrical potentials from the visual apparatus: (1) corneoretinal potentials; (2) potentials from various nerve structures, and (3) action potentials associated with muscle structures, of which there are several types, depending on the location of the electrodes. He describes his method for investigating the last type and shows an EMG from the medial rectus of a rabbit. He finds evidence confirming the origin of the EMG of the medial rectus from that muscle.

The term "electro-oculogram" (EOG) is employed by Franceschetti, Monnier, and Dieterle.[36] They find it provides an objective means for demonstrating oculomotor disorders. They show that comitant strabismus is a simple anomaly of position and that the EOG does not show pathological amplitudes when the squinting eye leaves the primary position, while in paralysis of the lateral rectus with retraction the EOG revealed a diminution of amplitude in the direction of the antagonist. In paralysis of gaze to the left, with paralysis of the left medial rectus, the abduction of the right eye produced an exaggerated amplitude and the amplitude to the return movement was diminished.

Dieterle and von Berger [37] point out the importance of electro-oculographic examination for the study of deficiency of an extrinsic ocular muscle. The object of their study was to find a standard placement of electrodes to allow equal potential variations in the horizontal and in the vertical movements. They report their method and the results on normal subjects. They were able to visualize a constant placement of electrodes for correct EOG investigation.

The electrical recording of eye movements has also been investigated by Mackensen and Harder.[38] Horizontal movements are best taken between the lateral canthus and the lateral orbital margin. With bitemporal leads the height of the measured potential is directly proportional to the extent of the deviation. This relation is very exact for movements up to 30 degrees. Only bitemporal

leads can be considered for quantitatively exact recording of horizontal eye movements.

No doubt many ophthalmologists long for the day when the diagnosis of motor anomalies will be made by a machine, but, judging from these papers, it is still a long way off. However, progress is unquestionably being made, and it is obvious that this approach to the study of ocular motility will be receiving increasing attention.

HETEROPHORIA ‖

Fixation Disparity.—A subject that is difficult for most of us and that has been restated for orthoptists by Ogle [39] is that of fixation disparity. He describes simple targets having identical figures, as well as dissimilar marks that are used to demonstrate this phenomenon. The identical figures are fused, but the dissimilar marks are displaced with respect to each other if there is heterophoria; i. e., they are displaced in the direction of the phoria. The basic fact is that the subjective visual directions of the monocularly seen details are different from the visual direction of the binocularly seen details. Fixation disparity must be due to fused images falling on disparate retinal elements. The eyes must be converging to a point in front of or behind the target. Monocularly seen details will appear displaced an amount equal to this disparity. Thus, it is an exhibition of phoria while the fusion of similar images is maintained. The magnitude of the displacement depends on the muscular imbalance, on the fusional compulsion reflex, and on the type and pattern of the details for fusion on the targets; the more fusion detail the smaller the fixation disparity. If the details are more in the periphery, the disparity is larger because Panum's areas are larger. The strength of the compulsion to fusion is greatest when the disparities are such that diplopia is imminent. Fixation disparity permits one to find the change of muscle imbalance while fusion and normal convergence and accommodation are maintained.

‖ See Jampolsky.[37]

Retinal slip is the expression that Marton [40] uses for fixation disparity. He also used a stereoscope to study it and measured the disparity with prisms, finding the same close relationship between the amount of prism and the amount of phoria. He divides heterophorias into (1) compensated, with no symptoms, and (2) uncompensated. Compensation is brought about by the action of the relative vergencies. These values will vary according to the amount of accommodation, the size of the test object, and the state of the accommodation-convergence relationship. A phoria without retinal slip is compensated. If the prism required to align the test objects is less than the measure of the phoria, the former is the amount of prism to be prescribed. This may explain why a partial prismatic correction is preferred in some cases. He raises the question: Is the amount of slip the true binocular imbalance and the heterophoria revealed by dissociation an artifact? He suggests that the measurement of the horopter may be of service in the study of heterophoria.

Divergence Insufficiency.—Lyle [41] has the impression that divergence insufficiency is on the increase and suggests that it may be due to certain brain-stem injuries from automobile accidents, virus encephalitis, and vascular lesions in the aged. He accepts the proof that divergence is an active process and believes that the divergence center should be considered as a more extensive "neural arc," or "relay station," through which are transmitted selected stimuli. He discusses the anatomy and possible location of lesions affecting divergence. He also lists the causes and differential diagnosis. After describing the typical features of divergence insufficiency, he calls it "manifest bilateral convergent heterotropia." If the diplopia persists after use of prisms or occlusion, he has found that shortening both lateral recti usually gives satisfactory results.

Convergence Insufficiency.—This condition is defined by Kent and Steeve [42] as prism convergence of less than three times the exophoria for near, prism convergence of

less than 15 D. for near, or a prism convergence recovery point of 5 D. or less. Of 4461 patients of military age referred to the refraction clinic, between 3.15% and 4.9% had defective convergence. Sixty per cent had headaches; 49%, blurring of vision; 34%, ocular fatigue, and 21%, occasional diplopia. Those benefited by orthoptic training were not numerous, but details of their methods are not given. Burian [43] disagrees with their conclusion in that he believes orthoptics to be of definite value in the vast majority. He suggests that convergence insufficiency may cause the patient to become neurotic, instead of the convergence insufficiency being a result of a neuropathic constitution, as is often thought.

After studying 67 cases of convergence insufficiency, Toselli [44] believes that an incomplete development of the convergence function is usually present. He bases the diagnosis on the receding of the convergence near point, a relative exophoria, and a decrease of prism convergence. The interpupillary distance, refraction, and aniseikonia may be contributory factors. It occurs more frequently in adolescent girls, and the first symptoms may appear during convalescence from a febrile disease, accompanied by mild and transient psychic disorders.

CONVERGENT STRABISMUS

Accommodative Esotropia.—The American Orthoptic Council and the American Association of Orthoptic Technicians have given us another of their excellent symposia, this time on accommodative esotropia. As in their previous symposia, their aim was not so much to bring out new material as to give an up-to-the-minute, authoritative summary of current knowledge on the subject. It was entirely fitting that the four speakers should all come from Washington, D. C., which most will agree is headquarters for the treatment of accommodative strabismus in this country. Almost by definition, these symposia are difficult to review because they are so factual.

The etiology and physiology are discussed by Parks.[45] Accommodation and convergence are in constant ratio, called the "*accommodation-convergence association reflex.*" This varies in different persons but is constant for each. A 1 : 1 ratio is the ideal for an emmetrope with orthophoria (accommodation in diopters; convergence in meter-angles). This ideal seldom exists; so adjustment of one or the other must be made (dissociation), which is performed through the *fusional vergence reflex.* Most persons use the accommodation required for clear vision, then adjust their convergence. Accommodative strabismus develops when the amount of accommodation for clear vision is reflexly associated with so much convergence that the vergence reflex cannot overcome it. An abnormal accommodation-convergence association reflex is identified by making the prism-cover measurements for distance and near with the accommodation controlled. An abnormal reflex that causes accommodative esotropia will have greater convergence for near. The fusional vergence reflex may cause accommodative strabismus by failing in its function: (*a*) fusional divergence adequate but not used because of fatigue or heterophoria, or when there is any barrier to foveal fixation; and (*b*) fusional divergence inadequate. *Relative fusional divergence amplitude* is the amount of divergence available while a specific amount of accommodation remains constant. Direct measurement is difficult because it is hard to control the accommodation. In measuring it indirectly, while maintaining constant convergence, the accommodation is increased until bifoveal fixation cannot be held, then reduced until it is resumed; the prism cover test will then give a rough measure. It is easily measured on a haploscope.

The clinical course and management are discussed by Costenbader.[46] He divides esotropia into three distinct groups: (1) constant deviation (nonaccommodative); (2) intermittent (accommodative), and (3) mixed. The accommodative group is divided into two types: (1) persons with excessive hyperopia and a normal accommodation-convergence ratio, and (2) persons with less hyperopia but an abnormal accommodation-con-

vergence ratio. The other three factors that determine the type are (1) adequacy of relative fusional divergence, (2) state of foveal vision, and (3) size of the basic heterophoria. In the first type, the deviation is eliminated by full hyperopic correction; amblyopia is not severe and responds well; correspondence is normal, but suppression is frequently found. In the second type, there is still a large esotropia for near with the full hyperopic correction, which is alleviated with added plus sphere (bifocals); the near point of accommodation is remote. For the purely accommodative cases, the treatment consists of improving the visual acuity with occlusion, proper glasses (the weakest correction that will allow bifoveal fixation and 20/20 vision under full cycloplegia, and the same for near, starting with +3 D.), and orthoptics (suppression overcome, diplopia encouraged, fusion stimulated, fusional amplitudes improved, accommodation and convergence dissociated). He does not use atropine because the blur encourages accommodative effort. Isofluorophate (Floropryl) has been encouraging, especially with an abnormal accommodation-convergence ratio. Surgery is seldom required—only if the basic esophoria is 10Δ or more and relative fusional divergence is inadequate.

The office training of these patients is covered by Tibbs.[47] She defines the goal of orthoptic treatment: the best possible visual acuity in each eye, and good binocular single vision by (*a*) dissociation and (*b*) proper harmonious relation between accommodation and convergence. She gives the actual exercises used to accomplish the steps enumerated by Costenbader. The home training is discussed by Kramer,[48] who feels that its value cannot be overemphasized and that it must be done daily. It consists of stimulating an amblyopic eye, antisuppression exercises with a red filter over the good eye (games, reading, physiological diplopia), fusional amplitude with the hand stereoscope, and dissociation of accommodation and convergence (stereoscope; Orthofusor, base in and base out; Walraven bar separator; minus fitovers). This symposium should be read in full by anyone who is interested in treating these patients.

Interesting observations in two cases of accommodative esotropia with divergent position of rest are made by Konstas.[49] The first case had straight eyes for distance. Under cover, one eye diverged 17 degrees; the other, 22 degrees. When he was accommodating, there was an esotropia of 14 degrees. His retinoscopy findings (without drops) are particularly interesting: (*a*) eyes divergent, +6 D; (*b*) eyes parallel, +4 D; (*c*) eyes convergent, +0.5 D, after atropinization, +6.5 D. With full correction, the eyes diverged, but the patient could hold the eyes straight and had good fusion with depth. Konstas' second case was similar but the patient was older, with amblyopia and anomalous correspondence. She showed the same differences in refraction with her eyes in the three positions. The treatment was to give the glasses found by retinoscopy in the parallel position, without drops. The author believes that a divergent position of rest and the absence of secondary muscle changes are the two requirements for the gradual transition from esotropia to exotropia.

Miotics.—The literature does not reflect the increased interest in the use of miotics in the treatment of strabismus. Although Abraham [50] has been the only one to write on this subject in this country, except for Costenbader's brief note,[46] a number of ophthalmologists are following his lead in using isofluorophate in the treatment of intermittent accommodative esotropia. This year Abraham [50] has described in more detail the most serious complication of this treatment, namely, the development of cysts on the pupillary margin. He found them in two-thirds of 66 cases. They appeared in from 1 to 40 weeks (average 10 weeks), and it is his impression that they are related to the degree of miosis rather than to the strength of the drug. The cysts disappeared in the majority in nine weeks after discontinuance of the drops, leaving shrunken tags. The cysts tend to re-form on resumption of the drug.

Similar cysts have been seen after the use of miotics for glaucoma. Swan,[51] speaking

only of glaucoma cases, reports that the cysts have increased in size and frequency with the use of more powerful miotics. He finds them more frequently in children, whose pupils react more intensively, and believes that the iris sphincter pinches the epithelium against the lens, with resultant fluid retention (Abraham agrees). Unfortunately, no histologic examination of these cysts has been reported. There is no proof that they do any harm unless they fill the pupil to a degree sufficient to reduce vision; however, they are a definite deterrent to the continued use of isofluorophate in strabismus cases.

One report on this subject has come from abroad (Switzerland). Huber [52] discontinued the use of atropine as a treatment for strabismus in small children because the attempt to see more clearly often increased the convergence (Costenbader [46]). Huber has now used miotics (physostigmine or Mintacol [diethyl-*p*-nitrophenol phosphate]) in 71 unselected cases. He found 0.5% Mintacol most satisfactory, given every morning. He has continued it for 12 months, without undesirable effects. He noted "blister-like distentions" of the pigmented pupillary edge in some cases. No effect was observed in 30 cases, while in 41 the angle was reduced more than 3 degrees. A strong effect, of over 10 degrees (maximum 15-20 degrees), occurred in 25%, in most of which there was hyperopia of 4 to 7 D. In other words, he confirmed our impression that the treatment works best in intermittent accommodative esotropia with onset at between 2 and 3 years of age, but the degree of hyperopia was somewhat higher in his most favorable cases. He found glasses and Mintacol to be of equal value in reducing the angle in about half his patients; in the others, Mintacol was superior. It was useful in reducing the angle in preparation for orthoptics and to remove small postoperative angles. He believes that an artificially produced spasm of accommodation is its chief effect and that it is harmless, although he advises discontinuing the drug from time to time to test the stability of the effect. Unfortunately, he judges the effect only in terms of reduction of the angle of deviation, and not in the reestablishment of binocular single vision.

Blind Spot Mechanism.—The blind spot mechanism was the subject of Swan's Gifford Lecture.[54] He did not find total suppression in a single case of 500 esotropias. Perception was monocular only in the central field of binocular vision. Two mechanisms make this possible: (1) suppression, or (2) the blind spot mechanism. In this lecture he reviews his previous work and adds considerable new material. He found the physiologic blind spot of the deviating eye overlying the point of fixation in 80 of 296 cases. The blind spot covers the fixation area in deviations of from 9 to 21 degrees; so it probably does so in an esotropia of 12 to 18 degrees. The area of monocular perception can be plotted with the Lancaster test. Swan recognizes four groups: (1) the blind spot syndrome, with no sensory abnormalities; (2) the blind spot mechanism with amblyopia; (3) the blind spot mechanism with anomalous correspondence, and (4) the blind spot mechanism with suppression. The blind spot mechanism can occur in large deviations partially corrected by surgery, and with pareses, chiefly in children with congenital paresis of a vertical muscle. He has treated over 200 patients, and most have developed comfortable binocular single vision when treatment could be completed. Elimination of the deviation is most important, or they tend to return to utilizing the blind spot. In about half the patients binocular single vision occurred spontaneously after successful surgery; the others required postoperative orthoptics. Prolonged preoperative training is not important. Patients with amblyopia and anomalous correspondence may use the blind spot mechanism but do not have the syndrome. Since amblyopia plays no role in the relief of diplopia, these patients may use the blind spot. The treatment is more difficult, and the prognosis is not as good. The amblyopia must be overcome before operation. The cases with anomalous correspondence were usually those with a large deviation partially corrected by surgery. The change in the deviation disrupts suppression,

and the blind spot relieves the diplopia. A new and harmonious angle of anomaly develops. They have made a high degree of sensory adaptation to the motor anomaly; they are asymptomatic and have a good substitute for normal binocular vision. Prognosis for binocular vision is poor, and even the cosmetic results are disappointing; yet Swan still advises occlusion, orthoptics, and surgery. The cases of the blind spot mechanism with suppression are not included, as they are to be the subject of a later paper.

Fritz ¶ believes that amblyopia neutralizes diplopia only at low angles. At 15 degrees, the second image is avoided because it falls on the blind spot. He seems to imply that most squints are in this group. The permanence of this false projection involves secondarily anomalous correspondence of the points which surround the papilla. Only very rarely does occluding the fixing eye serve to bring macular projection on a level in these cases. Reeducation of false projection generally leads to imperfect results. Reeducation should be done monocularly, using his striped glasses (described in 1952). Once the defect is reduced to simple amblyopia without anomalous correspondence, occlusion of the good eye is used to bring up the visual acuity. This rarely requires more than two months; he then operates, or the poor projection will return. Early surgery is imperative in anomalous correspondence as soon as macular function has been recovered.

Alternating Strabismus.#—Alternating tropia is defined by Laughlin and Stickle [54] as ability to fix at will, or unconsciously, with either eye and, at the same time, suppress to some extent the visual impulses from the nonfixing eye. The presence or absence of alternate suppression is the determining factor. The authors recognize three groups of alternators: 1. True alternators: It is impossible to demonstrate diplopia, complete suppression, and lack of simultaneous retinal perception (they prefer this expression to "no correspondence"). These cases are not amenable to active therapy, and surgical overcorrection should be avoided. 2. Alternators with abnormal retinal correspondence: There is a paracentral area of suppression (the behavior on the major amblyoscope is described), and simultaneous retinal perception in all areas outside of this. Alternate patching and orthoptics to break up suppression and establish normal retinal correspondence are important. Surgery is indicated after normal correspondence is established, and postoperative orthoptic exercises are used to maintain the normal correspondence. 3. Alternators with normal correspondence: The suppression area is parafoveal. These patients are the most likely to have good results. One can try for fusion before operation, but they are easily trained after operation. The development of fusion is much more difficult in alternators than in persons with monocular strabismus.

Bedrossian [55] divides all cases of alternating strabismus into cases of normal and cases of anomalous correspondence, including in the latter all cases in which the two foveas no longer have a common visual direction, regardless of the type of suppression pattern. Suppression is not related to correspondence and is almost always regional, rarely total. He believes that the work of Travers has disproved that total alternating suppression exists in alternating strabismus. Considering as alternators all patients having visual acuity within two lines' difference, he finds that they constitute 50% of squint patients and reports on 76 of them. Anomalous correspondence occurred in 64% of those appearing before the age of 3 years, and in only 20% of those with strabismus coming on later. He got good surgical results in 96% of cases of normal correspondence, and the alignment was maintained. Good results were obtained in only 61% of the cases with anomalous correspondence. All cases with anomalous correspondence detected by the after-image test showed the same anomaly on the synoptophore, but a few (4 cases) were normal by the after-image test and abnormal on the synoptophore. The only cases that became normal after surgery were in the

¶ Fritz, A., in discussion on Parfonry and Polliot.[63]

See Costenbader.[50]

last group (three cases). As in most reports of results, too many criteria are omitted and too few cases are given to permit real evaluation.

Mirror Writing.—With the observation that the leading authors on strabismus make no mention of the relationship to mirror writing, Corcelle [56] offers a preliminary report. Three cases are reported, all with marked amblyopia and all left-handed. One child, of 4 years, with his left eye squinting, wrote correctly with his right hand but demonstrated mirror writing with his left hand. The author speculates as to whether the strabismus, left-handedness, and mirror writing could all be caused by dominance of one hemisphere.

DIVERGENT STRABISMUS *

Jampolsky [57] has tried to give the differential diagnostic characteristics of intermittent exotropia and true exophoria. He attributes much of the confusion to lack of definitions, which he proceeds to supply. Intermittent exotropia is "characterized by a change in the relative position of the visual axes from parallelism to that of divergence, occurring unaware to a naive patient, without change in accommodation or in the subject of regard." For any given fixation distance, if the eye wanders out without diplopia, the patient has intermittent exotropia for that fixation distance. Exophoria is a wholly latent divergent deviation that is never manifest except by conscious awareness, and always with diplopia. Suppression is the essential differential point; there is hemiretinal suppression in intermittent exotropia. The author believes that these two conditions have a common etiology and that they usually arise during infancy. If profound hemiretinal suppression does not develop (before 6 years), it remains an exophoria. If suppression does develop, then it becomes an intermittent exotropia. So it is a problem of profound sensory change and continued motor change. There is probably an active factor in the divergence mechanism. Four points are important in the differentiation: (1) There may be intermittent exotropia for either distance or near (fixation at more than 6 meters is more likely to bring it out); (2) a fixation object requiring attention is less apt to bring out the full extent and the exact characteristics; (3) inattention brings out intermittent exotropia, and (4) some patients learn tricks of awareness of eye position. In exophoria there is no profound sensory adaptation; so it is a problem of motor balance: position of rest in relation to fusional amplitude. Any treatment (prisms, orthoptics, surgery) that even partially improves motor balance is effective in eliminating symptoms. In intermittent exotropia these measures fail. Special attention must be given to the sensory aspects, especially suppression. More than partial correction of the motor position must be achieved. No fault can be found with Jampolsky's ideas. It is only regretted that he could not put them down in simpler language.

In discussing exotropia with bilateral elevation in adduction, Urist [58] expresses the opinion that the vertical deviation is secondary to the dissociation of the eyes caused by the exotropia. He agrees with Scobee and Costenbader as to the early onset: 40% at birth, 80% under 5 years. With the cover test he found that 60% had a greater deviation for near, but by the corneal reflexes 78% had greater deviation for distance and none greater for near. The amount of the exotropia for distance was almost twice as large with the corneal reflex test as with the cover test. When the measurements are compared for straight up and for straight down, the exotropia was greater in 93% when looking up, with the cover test. He found a bilateral vertical deviation, with the cover test, in 68%; the right hypertropia was greatest up to the left and the left hypertropia greatest up to the right. By lateral versions, 73% showed bilateral elevation in adduction; this was marked in 43%. Urist considers this deviation a syndrome because of the following features: (1) bilateral elevation in adduction; (2) greater exotropia for distance; (3) greater exotropia in sursumduction, and (4) characteristic vertical deviations. He discusses the confu-

* See Costenbader.[50]

sion of classification, but, unfortunately, he does not succeed in dispelling this confusion.

In a second paper on the surgical treatment of this condition, Urist [59] reports the results in 54 cases. Bilateral recession of 7 mm. corrected the exotropia for distance and upward gaze without overcorrecting the previously straight eyes for near and down. It gave no limitation of abduction. The bilateral elevation in adduction disappeared, and the vertical cover measurements were much improved. Bilateral recession and a resection of one medial rectus are indicated where there is some exotropia for near. With the correction of the exotropia, bilateral elevation in adduction, wheel rotation, and alternating sursumduction, all improved, demonstrating that the deviations were probably secondary to the dissociation. This explains why bilateral recession of the inferior oblique alone will not correct the bilateral elevation. Urist finds a definite anatomic basis for recessions of the lateral recti up to 10 mm., without crippling action, thus confirming Scobee. Using Adler's diagram, he visualizes a functional equator of the globe relative to the orbital axis, rather than the anatomic equator and the optic axis. This functional equator would be 20 degrees posterior to the anatomic equator on the lateral side, or about 4 mm., thus permitting 4 mm. more recession before reaching the equator. (The same reasoning causes him to disagree with Scobee about recessing the medial rectus to the anatomic equator.) Bilateral recession gave functional cures in the majority of his cases. Bimedial resection, as a primary procedure, gave consistently poor results. He concludes that in exotropia with bilateral elevation in adduction there is apparently overaction of the lateral recti. His observations are very interesting, but it is not apparent whether he is discussing a very special type of exotropia, in which case he has had a most unusual experience, or is finding features in intermittent exotropias that the rest of us are missing. His justification of larger recessions of the lateral recti appears to be logical, and may be important, because evidence is slowly accumulating to indicate that they may be the best surgical treatment of intermittent exotropia even when there is a large deviation for near.

An interesting observation is made by Lance [60] that, while the British report the incidence of exotropia as between 4% and 11% of squint cases, in Sydney they find it to be 25%. She wonders if bright sunlight could cause the greater incidence. Since orthoptics can do little for constant exotropia, she concentrates her attention on intermittent exotropia. Cases of this type are divided into (1) divergence excess (intermittent exotropia for distance only), 25%; (2) convergence insufficiency, 19%, and (3) combined (primarily divergence excess, 27%; primarily convergence insufficiency 2%, unknown, 26%). The treatment is the same for all, but the diagnosis helps in the prognosis. The onset was under 6 years in 61%. Amblyopia was rare; 79% had 6/6 acuity in each eye. Sixty-nine per cent were females. Only 12% had no vertical deviation, and these all had convergence insufficiencies. Lance is not convinced that the abnormal binocular responses observed were those of true anomalous correspondence. In the treatment, occlusion is important; it was used in 74%. It relaxes the eyes to the static angle and breaks down suppression. Recognition of diplopia when the eyes are diverged is taught. Converging exercises are used after the patient can recognize diplopia with either eye fixing, can relax well, can hold simultaneous macular perception, and has fusion and stereopsis at the angle of deviation without suppression. Suppression must be eliminated, or there is danger of spasm of convergence. If operation is indicated, it should be done before the converging exercises, to avoid postoperative overconvergence. If orthoptics has failed, the patient has reocclusion and is given relaxing exercises before operation. This paper is an excellent discussion of the orthoptic treatment of intermittent exotropia. The author concludes that orthoptic treatment is usually all that is necessary to improve converging power in convergence insufficiency, but that in inter-

mittent exotropia and the combined types the patients continue to have a static divergent angle and surgery is the only hope for a permanent cure.

Some confusion in the surgical approach to nonparalytic exotropia is emphasized by the observation of Mulberger and McDonald[91] that 31 clinic patients received 40 operations, which included 13 different procedures. They classify their cases into intermittent, alternating, constant, and postoperative. They feel that orthoptic exercises are of great value, but if a definite exotropia exists, the child should have an operation before the age when he can cooperate intelligently for the exercises. The authors apparently mean objectives of surgery when they speak of indications and use the expression "binocular function" without defining it. All intermittent exotropias sufficient to cause symptoms or to be cosmetically noticeable require surgery, regardless of age. The authors base their choice of surgery on the ocular rotations. They usually do a bilateral recession to the equator, regardless of the degree. The surgery should be bilateral and symmetrical. An immediate slight overcorrection is desirable. For alternating exotropia (they do not say whether this is intermittent or constant) of low degrees, bilateral recessions are advised. Bimedial resections are used for higher degrees. In some cases, all four muscles are operated on, but not at one time. Constant exotropias are usually only cosmetic problems, with the deviating eye amblyopic, so that a resection of the medial rectus and a recession of the lateral rectus is the primary procedure. They have found it difficult to overcorrect in these cases. Postoperative exotropia is more difficult to correct. They agree with the generally held conviction that more surgery is necessary to correct it than was necessary to produce it. They usually work on the muscles originally operated on. They report the results of 15 types of operations done by a number of different surgeons on 147 clinic patients and 38 private patients. Excellent results (straight with fusion) were obtained in 11% of clinic and 18% private patients; good results (cosmetically straight: ±5 degrees of deviation) in 36% of clinic and 60% of private patients; improvement (less that 10 degrees of exotropia remaining) in 29% of clinic and 18% of private patients; poor results (greater than 10 degrees of exotropia) in 24% of clinic and 3% of private patients. This paper is of interest because it gives the judgment of two surgeons who have obtained remarkably good results in this difficult group.

In discussing an unpublished paper by Mulberger on the surgical management of intermittent exotropia, Krewson † gives us his ideas on the subject. He also prefers bilateral recessions, but in larger deviations he adds a resection of one or both medial recti. In later cases, with secondary convergence insufficiency and a tendency to fix with one eye, he does a resection and recession. If there is a vertical deviation of over 2△ he is in favor of correcting it. While orthoptics may be helpful, surgery is the method of choice. Some of us are glad to note that he believes that surgery is especially effective in a very young child who has not developed foveal suppression.

TREATMENT

The French Report.—Last year this reviewer expressed acute distress over the annual report of the Ophthalmological Society of Paris, under the authorship of Parfonry and Polliot. Anyone is entitled to one's own opinion, but to have the official report of this Society advocate the methods of 25 years ago seemed like a serious backward step that might retard the handling of strabismus, at least in France, for many years. While I would prefer to forget this report, there have been subsequent developments which deserve to be recorded. It is probably impossible for an outsider to get a true picture of the situation. The Ophthalmological Society of Paris is apparently second in importance only to the French Ophthalmological Society. Albert Poulard, on whose teachings the report is said to be based, was the author of a large textbook

† Krewson, W. E., in discussion on Mulberger.[92]

of ophthalmology, published in 1923, was the chief of one of the large eye services in Paris, and apparently was a distinguished teacher; but he was not well known in this country, and certainly not in the field of motility. (Only 40 pages of his 1457-page textbook were devoted to disturbances of motility, and half of these dealt with the surgical treatment.) He retired in 1935. Parfonry, the senior author of the report, had been one of Poulard's interns and had remained his devoted disciple. Parfonry died, very suddenly, before the report was completed; so that it devolved upon Polliot [93] to try to record faithfully the ideas of his confrere; this, judging from the remarks of several of the discussers, he succeeded in doing only too well. It also became his burden to present the report to the Society. An entire day was given over to the presentation and discussion of the report, the printed text having been distributed previously. Leading ophthalmologists from all over France and distinguished visitors from Switzerland and Belgium were present and joined in the discussion (Onfray, Hartmann, Favory, Dollfus, Sedán, Dejean, Sevrin, Valière-Vialeix, Halbron, Braun-Vallou, Paufique, Hugonnier, Fritz, Sourdille, de Saint-Martin, and Pigassou).[93]

I have gone into this background in some detail because it helps us to evaluate the report, and particularly to understand the discussion. Apparently, Parfonry was a popular member of the Society, so that several members (most notably Hartmann), who would otherwise have criticized the report, refrained from doing so. While this may be commendable from one point of view, it is most regrettable in terms of the education of younger ophthalmologists in France, as well as the standing of French ophthalomolgy in the eyes of the world.

Several discussers were in agreement with the report. De Saint-Martin derived great pleasure in finding confirmation of his ideas. To try to add support, he had analyzed some of his own records and had found 25% spontaneous cures, which he advanced as an argument against early operation. The average age for operation in his cases was 18 years. Valière-Vialeix approved almost entirely of the main ideas presented. He emphasized the advantages of the improved facilities of the large centers and posed the dilemma of the ophthalmologists practicing in the smaller cities as a choice between allowing vision to be lost permanently or operating under conditions that could be catastrophic. He chooses the former and believes that most practicing ophthalmologists will agree. While seeming to criticize several minor points, Onfray gives the impression that he is fundamentally in agreement.

Many more criticized the report. Since most of the ideas presented in disagreement are those that we consider obvious and well established, I shall only summarize them. Amblyopia is reeducatable, as proved by numerous successes (Halbron, Thomas, Sevrin, Fritz). The fixing eye can become amblyopic with occlusion; so there can be no macular lesion (Hugonnier). Many believe in the value of orthoptic training and use it routinely (Sevrin, Pigassou). Cooperation of the patient and the parents, both for occlusion and for orthoptic training, is not difficult to obtain if they are adequately explained and if the doctor really believes in them (Pigassou). A serious inferiority complex frequently accompanies strabismus (Braun-Vallou, Sevrin, Sourdille).

While all the critics disagree with the dictum of operation only at puberty for cosmetic reasons, there is considerable difference of opinion as to the exact age for operation. Hugonnier will operate between 18 and 24 months in certain cases. It is probably the only chance for these cases of early onset to acquire binocular vision, but he is not sure. It is not his routine because the examination is difficult and the surgical result must be perfect. Braun-Vallou and Halbron state that operation before the age of 2 years is exceptional. Fritz, Thomas, and Sevrin advocate early surgery but do not give ages. Braun-Vallou considers 5 to 6 years the most favorable age, while Halbron favors 6 to 8 years if reeducation is possible. Most

discussers agree with the report that operation should be delayed if amblyopia cannot be corrected, but Hugonnier will operate in these cases between the ages of 7 and 8 years if the deviation is severe. One gets the impression that "early operation" means at the age of 6 to 8 years, and of course that is early as compared with Parfonry's.

Several of the discussers express the belief that general anesthesia is no longer an obstacle or that the risk is certainly worth taking (Braun-Vallou, Thomas, Sevrin, Sourdille), but they require trained anesthetists.‡ Paufique did many operations under drop ether anesthesia without mishap, and the only fatality that he has heard of occurred with local anesthesia. He points out that if the dictum of the report were widely accepted, it would rule out the greater part of general infant surgery. Hugonnier and Thomas both find frequent indications for operations on the obliques.

Halbron objects to the dogmatic statements of the report and expresses the fear that they could give rise to lawsuits from dissatisfied patients. Similarly, Sourdille thinks that the criticism of general anesthesia and of postoperative reeducation is dangerous. Paufique expresses the fear that denying binocular vision and the methods of reeducation may undo all the efforts of those who have been fighting to get them accepted by Social Security.

The comments of Sourdille deserve special attention because he, like Hartmann, had been one of Poulard's interns and a colleague of Parfonry. He felt that this required him to express his criticism. He believes, and Paufique agrees, that under the influence of modern thinking and methods, Poulard would not have continued to deny amblyopia or its correction. He regrets the absence of a controversy on a scientific level because of Parfonry's absence. The postulates of the report cannot be accepted as conclusions, as no proof is offered, and they contradict scientifically established facts. He insists that verification of these postulates is required and that it is indispensable that the Ophthalmological Society of Paris or a complete assembly of the French Ophthalmological Society demands a counterreport which will put the ideas of Poulard in their proper place.

There were numerous comments on the surgical technique described in the report, but these are of doubtful importance. As is so often the case, many of the discussers failed to confine themselves to the report, but used the opportunity to present their own ideas on the treatment of strabismus.

Several of the discussers expressed their "complete opposition" to the report; yet their discussion seemed to fall short of total opposition, so that it fell to an outsider, Malbran,[64] to place the report in its proper light. His outspoken and highly articulate criticism appeared in the Society's *Bulletin* a few months later. He characterizes the report as "the most detestable contribution that has been brought to the study of strabismus in recent years." For a surgeon to fail to make every effort to reestablish and maintain visual function is to transform himself into a cosmetician or an artisan. All surgery must be based on the exhaustive study of each case. After paying homage to the many outstanding workers in the field of motility, in many countries, he proceeds to tear down, one by one, the unproved statements set forth in the report, by stating the facts that we have all come to accept as scientifically established. It is unnecessary to repeat his arguments here, as they are the ones that came to most of us in reading the report. His criticism was by far the most complete and satisfactory of any that was offered.

In summary, I am convinced that there are many right-thinking ophthalmologists in France, but I regret that more of them did not get up and speak more vehemently against this report. One still wonders why Parfonry was selected to speak for the Society. Sourdille, charitably, suggested that it was an attempt to conserve the best of the old ideas. From our point of view, the only possible good to come from it might be, as suggested by Fritz, that it has stirred up a

† See Henry.[88]

reaction among those who consider binocular vision a precious function. The only hope of counteracting the vast amount of harm done would be to have a counterreport by a higher authority, as demanded by Sourdille.

Orthoptics.§—It is probably not safe to imagine new trends from the literature of one year. However, the striking increase in both the quantity and the quality of the articles from central Europe (Switzerland, Austria, Germany, and Belgium) tempt me to conclude that an important change has taken place in the last few years. Apparently, the English deserve most of the credit, because Lyle is frequently cited and there have been prolonged visits by English orthoptists. We have already seen some of their papers on amblyopia and anomalous correspondence, and there are several more on orthoptics. They are characteristically long and detailed, in many instances, and most of them are enthusiastic, reporting far better results than we have learned to expect. Let us hope that this is not just enthusiasm for a new method. Some of their ideas are of considerable interest. Unfortunately, in almost no instance is sufficient detail given for an accurate evaluation of the reported results.

In a long article on the bloodless treatment, Hamburger [65] develops the theory that normal binocular vision falls between two extremes. At one end, there is strong monocular dominance, and at the other, horror fusionis with rapid competition. Most normals show a competition between the two retinas. Induced dominance exists to some extent in all normals, indicating that monocular dominance by no means stands for full predominance of one retina. Strabismus can be divided into two general groups on this basis: (1) monocular with amblyopia, a variation of monocular dominance, and (2) alternating strabismus with anomalous correspondence, a variation of rapid competition. He believes that accommodation, temporary pareses, or influences of central nervous character bring about strabismus and the sensory conditions are an adaptation. The essential steps in his treatment are as follows: 1. Corrective glasses. He insists on full correction, including the astigmatism. 2. Elimination of amblyopia. This can be done without occlusion only in school; it must be continued to the *14th year*. 3. Rehabilitation or reawakening of normal binocular vision. (*a*) Monocular strabismus: Binocular vision with all degrees of fusion comes spontaneously if vision is rendered close to equal and axes are made parallel by glasses or operation. (*b*) Alternating strabismus: a perfect type of vision, in many respects superior to normal binocular vision. He advocates early operation (after 5 years of age) and prolonged orthoptic training. On the assumption that horror fusionis is the leading motive for anomalous correspondence, the *squinting fovea* must be made amblyopic by prolonged occlusion, both before and after operation, to enforce dominance of the other eye. He bandages the *squinting eye* for 4 to 8 weeks after operation, in addition to orthoptics, for which he favors Lyle's method of kinetic stimulation, for 6 to 12 months. In spite of this unusual approach, he concludes that a true and full normalization of the binocular act of vision is rare even with the most careful orthoptic treatment, and in most cases we have to be satisfied if the eyes are parallel.

Evans [66] bases his binocular examination and orthoptic treatment on the Stereo-Orthoptor. He treats all cases of strabismus up to 20 degrees orthoptically. For the larger deviations, especially those with anomalous correspondence, an operation is usually necessary. He divides his cases into four groups, as follows: 1. Amblyopia with normal correspondence, 27%. All but three patients were completely cured by orthoptics. Amblyopia often continued to improve after fusion was established and the eyes were straight. The average age at the first visit was 6.3 years. Amblyopia is curable at any age if there is normal correspondence. He found that fusion can be built up after the 10th or 15th year. 2. Pseudomacular or paramacular fixation. (Either this portion of the article is thoroughly confused, or I have not under-

§ See Amblyopia, Anomalous Correspondence, Accommodative Esotropia, Exotropia.

stood it.) 3. Suppression scotoma with normal correspondence. This must be removed by training, then treated like the first type. He was able to overcome horror fusionis in some cases. Secondary deviations have been reduced considerably by postoperative orthoptics. Hypercorrections and hypocorrections can almost always be leveled off by orthoptics started a few days after operation. Functional and cosmetic recoveries are expected in cases with normal correspondence treated by combined surgery and orthoptics, regardless of age. 4. Suppression scotoma with anomalous correspondence. He found third-degree fusion in eight cases. He always attempts to develop normal correspondence by intensive training. If this is unsuccessful, there are two possibilities: 1. Anomalous correspondence is accepted and fusion built at the *subjective angle;* by orthoptics the objective angle is reduced until it corresponds to the angle of anomaly; unharmonious anomalous correspondence is transformed to harmonious; thus the deviation is somewhat diminished and the patient is given binocular vision despite the anomalous correspondence. 2. Strabismus operation: It is impossible to predict how the angle of anomaly will act. The angle of anomaly may remain constant, causing paradoxical diplopia. This is avoided by suppression or renewed deviation tending to approach the angle of anomaly or by a secondary deviation. There may be covariation of the angle of anomaly due to the altered objective angle, in which case the correction from operation will rarely be lasting. A third possibility is that normal correspondence may start again. Harmonious anomalous correspondence may contradict operation because it may exchange undesirable diplopia for a form of binocular single vision.

With the observation that there is no German book on orthoptics, Meitinger[67] undertakes to give a short outline, hoping greatly to increase its use. He has devised a simple, inexpensive instrument, called the orthoscope, which will shortly be on the market. It is based on the Pigeon-Cantonnet stereoscope, with three wings, which all turn freely on a common axis. There is a mirror on each side of the center wing and a picture holder on each side-wing which can be moved horizontally and vertically and rotated. There are three scales in degrees, for measurements. The pictures can be illuminated alternately. He follows Lyle's classification of anomalous correspondence but speaks of the "pseudomacula" and "pseudomacular correspondence," which would seem to be unfortunate terms. He describes the actual exercises carried out with the Orthoscope, following accepted methods. Apparently, he uses occlusion throughout the training period. In anomalous correspondence, if there is no success in 5 to 10 sessions, operation is usually indicated, with good prospects from postoperative exercises. Otherwise, operation is advised only after single binocular vision, with good fusion at the angle, is achieved. Orthoptic training is started as soon as possible after operation—1 to 2 days after a tenotomy, 8 to 14 days after a posterior displacement. He is convinced of the value of orthoptics. Unsuitable patients are (1) those with subnormal mental development, (2) those with suppression and anomalous correspondence too strongly fixed, in older patients, and (3) those whose parents are not convinced of the value.

The results obtained at the orthoptic school at Basel are reported by Rintelen and Hotz.[68] Their aim in the treatment of strabismus is to restore stereoscopic binocular vision. Operation, performed at the right time, is only one part of the treatment; its importance has diminished in favor of pleoptics and orthoptics. The results of amblyopia treatment are very good if it is carried out before the ninth year. However, stereoscopic vision was achieved in only a few cases. False correspondence is equivalent to binocular vision at the angle of strabismus, without stereopsis. They found false correspondence in 25% and consider the synoptophore more reliable than the red-glass or after-image tests. False correspondence is more predisposed to develop in the cases with a small angle. It does occur with large angles but tends to be inharmonious. False correspond-

ence can be treated only in a school. Their technique largely follows the English. They find few children ready for training before the age of 6, and by 8 their chances are becoming poor. Occlusion before the age of 6, as preparation, is important. False correspondence was eliminated in 34%, and one-half of these got stereoscopic vision. Results would be better if ophthalmologists would occlude earlier, especially in alternators. More frequent operation would also improve the results, particularly in small deviations, but the operation must be done early.

The impressions gained from the 147 cases examined during 10 months of orthoptic training at the Clinic Antoine Depage (Bruxelles) are given by de Lact[69]: 1. Orthoptic training is indispensable, for without it one ignores the degree of anomaly of binocular vision, and especially one knows nothing of one's ability to regain normal binocular vision. 2. Orthoptic treatment is almost always relatively short. For accommodative strabismus, the average was 11 sessions. In anomalous correspondence, normal correspondence often takes place at the first session, and satisfactory binocular vision is achieved in six sessions. Orthoptic treatments are not continued if there is no improvement in 6 to 10 sessions. The age from 3 to 6 is the most favorable. 3. Orthoptics is not a universal panacea, for many cases are not suitable. 4. Parents are surprisingly cooperative. 5. The orthoptists must cooperate with the doctor for good results, but the doctor can rarely do orthoptics. 6. Orthoptics offers an efficient way to combat strabismus and amblyopia, and is worthy of more effort.

The orthoptic results in 107 cases in which the treatment was finished are reported by Jonkers.[70] He found that binocular vision was disturbed in all cases of accommodative squint, but almost all the patients were completely cured by binocular training. Of cases of nonaccommodative convergent squint, he found anomalous correspondence in 50%, of which only 10% were cured. Of the cases with no correspondence (total suppression), 40% were cured. The cases with normal correspondence were of later onset, and 50% were cured. (He gives no details of the methods used, nor does he define a cure.) He is convinced of the value of pleoptic and orthoptic treatment.

In spite of the fact that both Great Britain and America have special orthoptic journals, fewer articles on orthoptics per se were published in 1954 than in recent years. Of particular interest is the paper by Lindsay,[71] in which, admitting that orthoptic treatment is not always satisfactory and that it often fails in going from the instruments to real life, he attempts to reassess present methods. Speculating on why animals do not squint, he decides that it may be a difference of intelligence, but that it is probably a difference in the mode of life. When an infant is presented with a conflict between what he sees and what he feels, either he learns to fuse and acquires full binocular vision (he learns to fuse through anomalous correspondence), or he suppresses one image. The author postulates that squint originates during the first few days of life and the initial effect is suppression. The deviation follows suppression and makes it easier, although the squint may not become apparent until later. The basic errors in present methods are these: 1. The child must be trained though the same stages in which he learns binocular vision normally: (*a*) simultaneous vision for color; (*b*) simultaneous vision for form; (*c*) simultaneous macular vision; (*d*) fusion, and (*e*) stereopsis. 2. Teaching by means of pictures has serious limitations; it should be correlated with tactile sense. 3. Too little use is made of hand-eye coordination. 4. Little or no use is made of physiological diplopia; appreciation of distance, hence binocular vision, depends on the interpretation of physiological diplopia. 5. A child is taught to fuse two separate pictures, an interesting trick. 6. Stereopsis is taught by pictures. The author outlines the requirements for a new technique: 1. There must be no interference with normal requirements for binocular vision; the instrument must allow the normal binocular field with associated monocular fields. 2. It must offer a means of eliciting diplopia rapidly and certainly. 3. The objects in the

binocular field must be appreciated as being single. 4. Physiological diplopia must be demonstrated and explained as early as possible. 5. There should be no attempts to separate binocular functions into color, form, etc. 6. There should be no fusion produced without physiological diplopia. 7. Emphasis should be placed on position. 8. Solid objects should be handled; they must be interesting. 9. It should be possible to exercise certain muscles. 10. Breadth of fusion must be exercised. The main requirement is that the brain must be educated first. The brain can be convinced through tactile sense; then the brain gives great assistance in directing the eyes and interpreting visual sensations. All visual phenomena must be associated with touch before they can be fully appreciated.

Lindsay proceeds to describe the apparatus that fulfills these requirements. It consists of a flat board with a septum at one end, which reduces the extent of the binocular field and produces right and left monocular fields. There are provisions for adding prisms, and it is arranged so that the cover test can be carried out at any time to check the fixation. Small lead animals are used that can be handled by the child. A squinter can obtain single vision somewhere, although it may be very close. The septum is removed when single vision is obtained; prisms are used if the angle is large. The various exercises are described, but no specific results are given. Anomalous correspondence is the main problem for further investigation.

Dobson [72] has been interested in anaglyphs and their use in orthoptics for a long time. She now describes the use of the Stereo-Motivator, which employs this principle and with which the two targets can be projected at any distance and are large enough to permit investigation of the peripheral retinas. There is no artificial infinity and no proximal convergence, as with the major amblyoscope. One must establish peripheral fusion of crossed and uncrossed pathways of both halves of the retinas in order to procure normal correspondence. She believes that the usual orthoptic method, on the major amblyoscope, of aiming at central fusion first, is putting the cart before the horse and conflicts with normal development. Peripheral fusion is much more easily obtained; peripheral correspondence and superposition prepare the way for central fusion. The details of the treatment are not given, and the only control mentioned is to ask the color of the pictures. We have learned to respect Miss Dobson's opinions, but she does not give enough details to enable us to evaluate this instrument.

In contrast to Dobson's ideas, and agreeing with Scobee that squint begins with foveal suppression and advances to macular suppression, Coates [73] believes that orthoptic treatment would be more successful if fusion training were done with targets that are so small that only the foveas are stimulated. He complains that with the present instruments there is little concern as to whether peripheral or central fusion is involved. He would like to have children brought for examination before the age of 2.

Some interesting conclusions as to binocular functions after squint operations are given by Law.[74] While based on a small series (40) of cases, they undoubtedly reflect his wide experience. Binocular results are almost never obtained when the deviation has existed from birth. It is very unusual to finish with a better level of binocular vision than the patient had at the start. Well-established anomalous correspondence is incurable. There are other reasons for amblyopia than disuse, as a certain percentage of patients fail to respond to treatment. Alternation does not materially affect binocular prognosis. Suppression, without amblyopia, is not a hopeless bar to binocular single vision. The earlier the operation the better for binocular vision. The degree of permanence of the result of an accurate operation is dependent on the binocular state. Orthoptic treatment is useless in removing the angle of deviation; it can only exercise, exploit, and facilitate the initial binocular state; it cannot improve it.

Janus [75] has seen cases harmed by orthoptics. There are two objectives in orthoptics that should tell whether training will be bene-

ficial or harmful: (1) to improve binocular perception so that diplopia is appreciated whenever the alignment is not correct, and (2) to develop the desire for binocular vision so that the amplitude of fusion is increased and any minor deviation overcome. Orthoptics is contraindicated in the following conditions: (1) a sensory anomaly in which true binocular vision is unlikely to develop (binocular stimulation may produce intractable diplopia), and (2) a motor anomaly in which it is unlikely that the axes can be made straight by orthoptics or surgery. Examples are the essentially alternating type without symptoms, older patients with postoperative diplopia, and defective binocular vision associated with anisometropia or aniseikonia. Suppression is a protective mechanism, so that we must be sure of a good surgical result before disrupting it. The author warns against prolonged occlusion in a nervous child. He believes that electroencephalographic records may give a reliable guide to the benefits to be expected from treatment.

The cases of 1000 patients with nonintermittent strabismus who were discharged in 1949 and reexamined three years later have been analyzed by Lynn.[76] A functional result was not obtained in 67%, and she asks why? There is no known method for breaking down anomalous correspondence; she has cured only one case; so she is satisfied with two good eyes and a cosmetic cure. She believes that there is an absence of fusion in the cases of early onset and does not try to develop it. Intractable amblyopia is another reason for failure. She considers boredom the chief obstacle to orthoptics. She has seen several children who were getting nowhere with orthoptics improve by getting out and playing ball, etc., using what she terms "outward projection into space," rather than the optical space used in orthoptics.

From a study of 74 cases of intermittent and accommodative esotropia, Luhr and Schlossman[77] found a reasonable chance for a nonsurgical cure when (1) the onset is between 4 and 6 years, (2) the vision is relatively equal, (3) the hyperopia is between 3 and 5 D., and (4) there is a well-functioning fusion faculty.

A small series of cases (37 with only orthoptics, 9 with preventive and postoperative orthoptics, and 6 with only postoperative orthoptics) enable Offret and Lombard[78] to conclude that orthoptic treatment is fully justified. Yet they raise the question, "Must we go further and say that it justifies early intervention?"; but they fail to give a direct answer. They believe that each strabismus case poses a human problem which the surgeon must solve in his soul and his conscience.

Stereopsis.—Increasingly, particularly in Europe, stereoscopic binocular vision is stated as the ultimate goal of the treatment of strabismus. Yet there has long been doubt as to whether stereopsis can be trained. The advent of 3-D movies has created problems for children with strabismus who have previously experienced no difficulties in their daily activities. Lindner[68] raises the question as to whether the strabismus cases that have been "cured" by orthoptic training can see these films correctly. Rintelen replies that they have correct stereopsis but that home exercises are necessary to stabilize it. Parkhill[79] believes that it should be the goal of orthoptics to enable these children to utilize 3-D movies, suggesting that the movies can serve as an aid to visual training. After listing the monocular clues for stereopsis, she gives the binocular requirements: normal correspondence, stability of fusion, absence of suppression, and bifoveal fixation. This paper is a good survey of stereoscopic vision, but she does not tell how the orthoptist uses it, nor does she answer the question as to whether it can be trained. Dobson[72] uses anaglyphs to train stereopsis, and Lindsay[71] believes that it can be taught through pictures.

SURGERY ‖

Age of Operation.—This is difficult to assess because "early" can mean anything from 1 to 7 years. A number of authors advocate early operation without giving the

‖ See Divergent Strabismus.

actual age. James [12] sees a recent change to early operation in Belgium. Thomas advises early operation, as soon as other measures fail, to permit normal development. Law [74] says the sooner the better for binocular vision. Others give definite ages. Costenbader [80] operates at the age of one year when it is indicated. De Jaeger and Bernolet [6] favor operation before 3 years in order to allow the eye to develop macular function. Hamburger [65] advises operation after the fifth year if there is no progress from orthoptics. Jonkers [70] waits until the child is old enough for training. Dejean [63] thinks that age is of little importance but waits until local anesthesia can be used.¶ In addition to those supporting the French report, there are still a few who are opposed to early operation, that is, by the age of 7. Lynn [76] holds this opinion because she cannot be sure that the axes are really aligned, anomalous correspondence may already be present, the parents may stay away if the eyes look straight, amblyopia may develop undetected, and there is risk of later deviation.

Choice of Operation.—This covers a wide range. Evans [66] advises operation for 20 degrees or more and usually does a tucking, according to the method of Bangerter, and a guarded tenotomy on the squinting eye. When there is normal correspondence, he aims for parallelism in one operation. If the deviation is over 20 degrees, Hamburger [65] prefers to operate on both eyes. He apparently likes a recession but points out the disadvantage of not being able to adjust it after the sutures are tied. For an alternating strabismus he does a 5 to 7 mm. recession of the medial rectus of the fixing eye, a tenotomy and setting forward of the lateral rectus of the squinting eye, and a controlled tenotomy of the medial rectus of this eye, aiming to overcorrect slightly. With a functional cure in mind, Lynn [76] usually does a resection and recession, but as this tends to limit dynamic convergence and the range of adduction, she has a tendency to reduce the amount of recession and increase the amount of resection.

James [12] reports that a bilateral resection is the most popular operation in Belgium, that it gives uniformly good results in all types of convergent strabismus no matter what the angle, and that there is no risk of postoperative limitation of the medial rectus and no impairment of convergence. This sounds almost too good to be true.

Dejean's [63] method of operating has given satisfaction for over 20 years. It can be regulated to apply to all cases of strabismus. He does a resection in which the suture includes a large surface of conjunctiva to avoid cutting the muscle. He shortens the tendon 1 mm. for 4 degrees. The antagonist is weakened by alternate transverse cuts. The tightening of the sutures regulates the position of the globe. He operates on only one eye and bandages both eyes for 10 days. This same operation is the routine used on Sedán's [63] service. Braun-Vallou [63] prefers a recession of the medial rectus, as it assures the integrity of convergence. She feels that the technique is of little importance. The operation should reestablish parallelism and give the muscles the maximum chance to function normally. Dollfus [63] uses a controlled tenotomy and does not bandage the eyes, in the belief that if the eyes are straight on the table, they should be left to function parallel.

In a general discussion of the principles of strabismus surgery, Costenbader [80] gives two objectives: 1. Binocular single vision, which presupposes adequate vision (at least 20/50) and must have accurate binocular alignment the major part of the time. Fusion is a major factor in maintaining this alignment. 2. The best possible alignment for cosmetic effect when binocular single vision is impossible. As to the time for operation, he believes that it should be done as soon as the parents are convinced and the doctor is certain enough of his findings to plan intelligently. Congenital strabismus should be operated on at the age of one year, but he delays if the deviation is variable. Variable cases of later onset must be treated conservatively. His indications for operation follow. For esotropia: (1) constant esotropia after comitance is established and the accommoda-

¶ See the French report.

tive element eliminated; (2) partially accommodative esotropia (only the nonaccommodative element should be corrected or secondary exotropia is almost invariable), and (3) the basic esophoria in some cases of accommodative esotropia (this may be safely operated on if over 10 D.) For exotropia: (1) constant exotropia; (2) intermittent exotropia, if 20 D. or more at 20 ft., and (3) exophoria, if over 20 D., if associated with convergence insufficiency, or if producing symptoms. When there is a vertical element, a functional cure is not often obtained. The results of surgery for vertical deviations are frequently less satisfactory than the original condition; yet if the hypertropia is cosmetically unsatisfactory, surgery is indicated. When a horizontal and a vertical element are combined, he operates on the larger element first. After describing his technique for doing a recession and a resection, he places these limits for the amount of surgery: recession: medial rectus 3 to 4.5 mm., lateral rectus 4.5 to 5 mm.; resection: medial rectus 6 to 7 mm., lateral rectus 10 to 12 mm. (see Cushman and Willard [83]).

Last year, Costenbader made a convincing plea for symmetrical surgery. This year, several authors have advocated the same thing. Costenbader and Bair [81] have now added considerable weight to this argument. Since most patients with strabismus do not have preexisting gross paresis or incomitance, they believe that surgery should attempt to preserve this comitance. Whether it is monocular or alternating is determined by dominance, refraction, and amblyopia rather than by muscle strength or weakness. They report 535 cases of nonaccommodative and partially accommodative esotropia and 131 cases of exotropia. They include only cases that were comitant before operation, i. e., a difference in the measurements in the two lateral fields of 10 D., or less. Postoperative incomitance is judged by obvious limitation, by difference in the primary and secondary deviations, and by a difference in the measurements in various directions. They believe that this will limit the opportunity for binocular single vision. Monocular surgery is advised for deviations of small degree when there is marked amblyopia, when the measurements are markedly variable, or when there is manifest incomitance. The fixation pattern had little influence on the decision of monocular or bilateral operation.

Binocular simultaneous symmetrical operations were done 299 times in 266 cases of esotropia: 239 bimedial recessions, 47 bilateral resections, and 13 bimedial marginal myotomies. One hundred ten binocular simultaneous symmetrical operations were done in 102 cases of exotropia: 74 bilateral recessions, 4 bilateral tenotomies, and 32 bimedial resections. Postoperatively, of the esotropias, 96% were comitant and 4% incomitant; of the exotropias, 98% were comitant and 2% incomitant. When the symmetrical surgery was done in two stages, 53% were comitant after the first operation, but 82% became comitant after the second. Of cases having only monocular surgery, there were 231 cases of esotropia, of which 48% were finally comitant (unilateral recession alone, 58% comitant; resection and recession, 39% comitant). They next studied the influence of the fixation pattern on the postoperative comitance. As alternators, they include spontaneous alternators and those with equal refractive error and equal visual acuity. This includes most congenital esotropias, most intermittent exotropias, and all "true" alternators. Of 665 patients, they found 241, or 36%, alternators. In terms of postoperative comitance, binocular surgery gave identical results in the two groups, but monocular surgery gave a little better results in the monocular group. While they admit that postoperative incomitance is not a bar to binocular vision, their figures indicate that the patient with comitance has a definitely greater chance for eventual cure. In congenital strabismus, of the comitant cases, 54% were straight with normal correspondence, while of the incomitant cases only 31% achieved this goal. In those of later onset the figures were 68% and 37%. These results seem entirely logical; yet two sets of their figures demand comment. While monocular surgery was advised only for small

deviations and for marked amblyopia (and not even all of these), of 535 cases of esotropia, 269 had initial monocular surgery. This would seem to indicate a surprisingly high number of cases in these two categories. The other figure that appears unusual is their finding of only 36% alternators. Since most patients with exotropia are alternators, this would indicate a surprisingly high number of monocular esotropias. Possibly their criteria for alternators are stricter than most.

A modification of the recession operation that has been used at the University of Oregon in over 500 cases is described by Swan and Talbot.[82] They review the anatomy of Tenon's capsule and its relation to the bulbar conjunctiva, the muscle capsule, the intermuscular membrane, and the check ligaments. Believing that Tenon's capsule forms the natural bed for the bulbar conjunctiva, they object to the way that the usual recession leaves exposed sclera, anterior to the muscle, to which the conjunctiva becomes firmly adherent. There is also danger that retracted Tenon's capsule may form scar tissue deep in the fornix that will nullify the recession, and that the check ligaments may be seriously altered. The essential steps in their operation are a vertical incision in the conjunctiva close to the semilunar fold, a horizontal incision in Tenon's capsule over the center of the tendon, and incisions in the intermuscular membrane along both edges of the muscle. After the recession, for which they use two single-armed 4-0 mild chromic sutures, locked in the tendon, Tenon's capsule is closed with two sutures with buried knots and the conjunctiva is closed separately with interrupted sutures. Scarring is minimal, and complications are less. The effect obtained is the same as with the usual recession, but the results have been more uniform. The same handling of Tenon's capsule is applicable to resections. As has been said by many authors, small variations in technique are probably of little significance, but this method of handling Tenon's capsule seems important. Other surgeons (notably Costenbader) have been using a similar technique, but this is the first time that it has been described in detail, with excellent illustrations.

Believing that private patients give more regular attendance and carry out home exercises more conscientiously, and that the records are more uniform, Law [74] has analyzed 40 unselected operative cases of comitant squint. For lengthening a muscle, he prefers a recession with absorbable surgical (gut) sutures. A full recession to the equator (8 mm. behind the insertion of the medial rectus) should give about 15 degrees of correction. Small recessions of 3 to 4 mm. are of no use. For shortening he does an advancement, believing that there is no advantage to a resection, since the sutures pull through the muscle to where the tendon begins, giving variable results. The advancement can be accurately controlled. He does not use an advancement alone because of the enophthalmos produced. A single recession is done for 15 degrees or less. A maximum advancement, up to 1 mm. from the limbus, gives 25 to 30 degrees of correction (including a recession of the opponent?). He has not been bothered by a raised area under the conjunctiva.

A carefully planned program for surgery, stressing minimum trauma, is advocated by Cushman and Willard.[83] They are opposed to stripping the capsule from the muscle and complete severing of secondary attachments. They have established the following maximum and minimum amounts for operations:

Recessions: Medial rectus, central tenotomy to 3 mm.; lateral rectus, 4-7 mm.
Superior rectus, central tenotomy to 4.5 mm.
Inferior rectus, central tenotomy to 10 mm. +
Superior oblique 5-9 mm.; inferior oblique, 2-4 mm.

Resections: Medial rectus, 3-6.5 mm.; lateral rectus, 6-10 mm. +
Superior rectus, 4-6 mm.; inferior rectus, 2-7 mm. and advancement
Superior oblique, tuck of 10-20 mm.
Inferior oblique, 4-5 mm. and advancement of 8-10 mm.

The maximum amounts are for corrections of 35 to 50 D. (or Δ).

They report 68% satisfactory results in 137 cases in which these rules were followed. These results are difficult to evaluate, but some comment on the limits that they impose is unavoidable. Obviously, a central tenotomy is not a recession, and it is only confusing to combine these terms as they do. A central tenotomy of a medial rectus of 3 mm. would hardly seem worth doing; yet they emphasize this limit particularly. On the other hand, a central tenotomy of an inferior rectus of 10 mm., if it is possible to do this much, should be a very dangerous amount by which to weaken a depressor. A 4 mm. recession of an inferior oblique is, again, of doubtful value. Their limit for a resection of a medial rectus is low, while that for the inferior rectus is inconsistently high. I believe that they are alone in advocating a recession of the superior oblique. This article strikes me as a beautiful example of how difficult and dangerous it is to try to tell anyone how much surgery to do.

A shortening and advancement of the less efficient muscle is advocated by Foroni.[84] He divides the tendon and the first layer of the muscle into halves, fastening them firmly to the limbal region. The two stumps of tendon are left protruding from the conjunctiva for several days to prevent slipping of the sutures. After a week, in which he believes that the advanced muscle is strengthened because of the opposing muscle, a recession of the opponent is done.

A new operation to this reviewer is that of "transposition of the globe," ascribed to Grimault by Thomas.[85] Incisions are made over the medial and lateral recti. A suture is carried through both edges of the lateral rectus tendon, beneath the conjunctiva to the nasal border of the superior rectus tendon, then to the upper edge of the tendon of the medial rectus, and over the tendon and conjunctiva to the lower edge of the tendon; after passing through this edge, it is carried subconjunctivally to the nasal edge of the inferior rectus tendon and back to the lateral rectus, and tied, thus completing a circle. The medial and lateral recti are tenotomized. The stump of one of the recti is grasped, and the globe is moved until it is straight; this is checked with the Maddox rod with the patient sitting up. Thus an advancement and a setting-back are accomplished, but the muscles remain in their *équilibre tonique antérieur*. Small vertical adjustments can be made. The amount of correction that can be obtained is limited to about 25 degrees of convergence and 15 degrees of divergence.

Being concerned with patients who are to wear contact lenses after a strabismus operation, Sarwar [86] has devised a technique that removes conjunctival scarring from the cornea as far as possible. He makes the incisions at the canthi. A resection is done in the usual way. A recession is not possible; so he lengthens the muscle by a halving technique. He starts motility exercises the next day and continues them as long as there is edema. He has had uniform success but admits that his method is more difficult.

Giardini and Rizzo [87] use a cataract mask for 7 days, starting 24 hours after the operation; this permits the eyes to look through narrow slits. They believe that the mask has little effect after resections, but after recessions the residual angle remained the same or was improved in 63% of cases, while without the mask this occurred in only 33%.

Stimulated by the French report, Henry [88] discusses the question of regulation of the strabismus operation. He aims his comments at his young confreres. He believes that it is impossible to regulate the operation on the table because of the preoperative medication and the anesthetic. He objects to the practice of adjusting threads one or two days after operation because it violates surgical principles, there is danger of infection, and it is impossible to see the eyes properly during this period in many cases. He emphasizes that the only way to regulate the operation is to avoid adhesions by minute care in the operation and by early postoperative movement. The operation must respect the surgical planes and facilitate the normal play of the capsule, the muscles, and their tendons. A tenotomy of the medial rectus is to be avoided, at all cost. The operation should always be bilateral because the paretic ele-

ment is bilateral and a dynamic correction to be obtained. The unilateral operation always produces an asymmetry. He prefers the capsulomuscular advancement, in which the capsuloconjunctival plane is never separated. He strives for an overcorrection. He stresses the role of the bandages, which are removed in 48 hours. If there is an overcorrection, he starts motility exercises immediately, repeating them two or three times a day for 10 minutes. These consist of moving the eyes up, down, and to both sides with increasing rapidity, and of convergence. The hypercorrection rapidly disappears. If the eyes are straight, he instills atropine and penicillin and replaces the bandages for a week. An undercorrection is an error in the operation. He believes that the postoperative exercises reestablish the normal physiological function in playing on the strength of convergence, which must never be weakened.

Anesthesia.—While anesthesia is hardly within the province of this review, the report of the Ophthalmological Society of Paris has made us wonder what is wrong with this practice in France. It is therefore reassuring to find the article by Willot and Leurent [89] justifying early squint operations and giving the details of their method of anesthesia. After deciding that tracheal intubation was the safest, they concluded that it was too dangerous; so they now follow the method used at Moorfields. The child is put to sleep with secobarbital (Seconal) or pentobarbital (Nembutal). Administration of the anesthestic is started with a mask, then shifted to an oropharyngeal cannula, using nitrous oxide, oxygen, and trichloroethylene.

PARALYTIC STRABISMUS

In the Doyne Memorial Lecture, Lyle [90] gives a detailed discussion of the factors which affect the prognosis and treatment of ocular palsy. The aim in any case is to restore comfortable binocular single vision over as large an area of the central and lower binocular visual field as possible and to make the movements of each eye as symmetrical and equal as possible. The chance of achieving this goal depends on (1) the previous existence of binocular single vision and (2) the existence of an effective range of ocular movements in each eye. Few patients with paralytic strabismus cannot be given a relatively normal ocular appearance in the primary position. He reports 229 cases with restoration of binocular vision over the most important part of the binocular field, of which 135 were congenital, with diplopia in the surprisingly high number of 93, and 94 were acquired. In the congenital group, the muscles were involved as follows: superior rectus, in 59 cases; superior oblique, in 39; lateral rectus in 17 (bilateral in 3); inferior oblique, in 6; inferior rectus, in 5; medial rectus, in 3; third nerve, in 3; superior rectus and inferior oblique, in 3, and superior rectus, in 1. The etiology of the acquired paralyses was nerve injury, in 57 cases; orbital injury, in 10; endocrine, in 11; vascular, in 4; encephalitis, in 3; meningitis, in 1; ocular myopathy, in 3, and unknown, in 5.

Lyle considers that congenital cases that are compensated, or that have become decompensated, have a good prognosis, with operation. The noncompensated, or constant, cases usually have a poor prognosis. In the acquired cases, diplopia is much more definite, compensation is unusual, and suppression is difficult. They may become decompensated, with partial recovery. The patient should be warned that more than one operation may be required. Several operations are better than the risk of an overcorrection. An undercorrection is preferable, especially in congenital paralyses. Binocular vision is not impossible in a partial third nerve paralysis; the operation is chiefly *weakening the corresponding muscles of the sound eye*, i. e., recession of the superior and lateral recti, and maybe the inferior rectus. He accepts Spaeth's explanation of the retraction syndrome. He prefers a recession of the medial rectus of the *sound eye*, to obtain a more symmetrical result. He has found the medial rectus similarly affected and believes that Brown's superior oblique tendon sheath syndrome is really a similar involvement of the inferior oblique. For a complete third nerve

paralysis, he did seven operations: operation for left ptosis; recession of right lateral rectus; tenotomy of left lateral rectus, plication of left medial rectus; plication of right medial rectus; fascial graft for the ptosis; tenotomy of right lateral rectus and resection of right medial rectus.

Out of 12,500 eye patients, among military personnel in East Africa, Taylor [91] found 38 cases of paralytic squint, or 0.3%. This was twice the incidence reported at the Glasgow Eye Infirmary. Abnormal head postures occurred in 13. Presumptive causes were direct trauma, seven; indirect trauma, seven; infection, seven; psychosomatic factors, two. The known results of treatment were too few to be significant.

Tendon Transplantation.—Two articles on tendon transplantation were missed last year. Hildreth [92] disagrees with Spaeth that the obliques explain the abduction obtained. He believes that the operation shifts the superior and inferior recti from the nasal to the temporal side of the vertical axis, so that adducting power is changed to abduction. This opinion is based on the following experiment: The medial and lateral recti were tenotomized. A suture around both the superior and the inferior rectus was carried around the lateral side of the globe. When this suture was tightened, displacing these vertical recti laterally, abduction was possible. In making the transplant, he confines the split to the tendons, making it only about 6 mm.

A case of traumatic bilateral sixth nerve paralysis is reported by Riffenburgh.[93] He did the following operations: (1) 10 mm. resection of both lateral recti; (2) 5 mm. recession of both medial recti; (3) 6 mm. resection of the right lateral rectus; (4) 6 mm. resection of the left lateral rectus. There was still an esotropia of 18 to 20 degrees and no abduction of either eye; so he did a tendon transplantation on the left eye and advanced the lateral rectus 3 mm., obtaining 15 degrees of abduction. The same operation on the right eye gave the same result. There was fusion except in looking well to either side or in extreme, downward gaze, and it was the same a year later.

François [94] transplanted strips from the vertical recti, with or without resections of the lateral recti, to obtain satisfactory results in five cases of congenital and acquired paralysis of the lateral rectus. The same method can be used for paralysis of other muscles, as was also suggested by Hildreth.

Surgery of the Obliques.—Another paper missed last year was that by McGuire [95] on surgery of the superior oblique. In inferior rectus paralysis, when the paretic eye is used for fixation and the paralysis is not marked, he favors weakening the opposite superior oblique, for which he prefers Berke's tenotomy. He believes that paralysis of the superior oblique is the commonest vertical paralysis and that "it produces by far the most distressing symptoms of any ocular motor weakness," because of the downward field of action and torsion. He describes his variation of Wheeler's operation for advancing the superior oblique tendon, which is actually a resection of the tendon, resuturing it to the insertion. In paresis of the superior oblique with the sound eye fixing, he advocates shortening the tendon. If the paretic eye is fixing, he prefers the same operation combined with recession of the opposite inferior rectus if the deviation is large. He finds two disadvantages to Wheeler's operation: (1) It may not hold, and (2) the new insertion is dangerously close to the macula. McGuire's operation has been criticized because it involves tenotomizing the superior rectus. He has done 65 cases, which seems like a remarkably large number and makes us wonder if we are missing these cases.

Fink [96] has given a characteristically thorough discussion of this same subject. He finds a trend toward confining the surgery to the field of the involved muscle. He believes that this changing attitude is due to (1) the importance of superior oblique defects, and (2) more accurate diagnostic criteria. Failure to recognize the superior oblique factor may cause a failure in correcting the horizontal deviation. The rule for

correcting the horizontal is not the same in the presence of a vertical. The vertical deviation should be corrected as early as possible; yet he cautions against overcorrections and points out that a given amount of surgery is more effective on the vertical than on the horizontal. In the majority, the ultimate correction is not expected from one operation. He usually operates on both eyes when there is a marked deviation. He saves the vertical recti for a secondary step and places 5 mm. as the limit for a resection and 3 mm. for a recession (Cushman and Willard [83]). If the superior oblique is underacting, it should be strengthened; if overacting, it should be weakened. The surgery should be confined to the reflected tendon. For overaction he does a tenotomy or a tenectomy, grading the effect by the amount of tendon removed and the closeness of the pulley. For underaction he finds the tuck most satisfactory and had devised an instrument for this. He believes that it is simpler and safer, that the effect can be more accurately graded, and that an overeffect is easily remedied. It is not necessary to cut the superior rectus. The minimum tuck is 5 mm., giving 5 to 7 degrees of correction; the maximum is 10 mm.

Two comments from papers reviewed above are added here for completeness. Costenbader [80] considers a recession of the inferior oblique a more exact surgical procedure than the myectomy but does not believe that its superiority has been proved. He does a myectomy if no operation on the lateral rectus is indicated. Law [74] operates on the inferior oblique for an "overelevated eye in adduction" and does a myectomy through the skin.

Ocular Torticollis.—Calmettes, Deodati, and Pigassou [97] define ocular torticollis as an abnormal position of the head caused by a disturbance of ocular motility, usually a paralysis of a vertical muscle. They present eight cases, of three types: 1. Congenital torticollis with absence of fixation of one eye. Surgery and visual reeducation lead to disappearance of the torticollis and the acquisition of stereoscopic vision in all cases. 2. Congenital amblyopia with strabismus; this is not correctable. 3. Binocular vision with head tilt but vertical diplopia when the head is straight. They urge early treatment. All of their cases made a total recovery of vision with stereopsis after operation and orthoptics.

Retraction Syndrome.—Cases of retraction syndrome are still considered to be of sufficient interest to be worth reporting. Four such reports appeared during the year (Magni,[98] Modrin,[99] Alojmo,[100] Stanković and Stanković [101]) that add little to our knowledge of these cases (Lyle [90]).

VERTICAL AND HORIZONTAL DEVIATIONS COMBINED

The importance of understanding the anatomic and physiologic differences between vertical and horizontal mechanism is emphasized by Fink.[102] Stable vertical balance is essential to the binocular act. The vertical mechanism has a very limited range of reflex power of adjustment. Even a small disruption of the vertical balance produces a definite mechanical obstacle. With his usual thoroughness he proceeds to list the peripheral motor defects, which are too numerous to include here. Of particular interest is his explanation of overaction of the inferior oblique in adduction. He believes that a congenital maldevelopment in the planes of action of the oblique muscles gives a logical explanation. In evolution the superior oblique has lost power.

> "The resulting physiologic imbalance of opposing obliques coupled with an appreciable imbalance in the planes of action of the obliques in their relation to the anterior-posterior vertical plane of the globe may produce an upshoot or abnormal action of the inferior oblique."

This may also explain some convergence and divergence anomalies.

A vertical component was found in 44% of 432 cases of comitant strabismus by Mattos and Blois.[103] They advocate early treatment to avoid spastic contracture of the eye and neck muscles. They correct the vertical component when operating for the lateral by suturing the medial and lateral recti higher or lower.

In a short article for orthoptists, Crone [104] discusses this subject. The great majority of deviations are nonparetic upshoot in adduction and dissociated hypertropia (occlusion hypertropia, called alternating hyperphoria if bilateral). There is a definite relation between alternating hyperphoria and upshoot in lateral gaze as they are frequently seen in combination. He discusses the various theories, none of which have been proved. The prognosis for orthoptics and binocular vision is poor (Crone [109]).

Experimenting with normal subjects, Venturi [105] found that at the limit of vertical fusion there was marked reduction of the amplitude of horizontal fusion. In patients with hypertropia, correction of the vertical with prisms was followed by disappearance of "all trouble" and changes in the amplitude of fusion and cyclophoria. He believes that his findings prove the importance of correcting the vertical deviation.

In 100 patients with horizontal tropias, Focosi and Muscas [106] found 53 measurable vertical deviations. They also found that the vertical does not disappear after an operation to correct the horizontal. Verticals occurred in 54% of the 79 cases operated on for comitant strabismus that were studied by Giardini and Rizzo.[107] After recessions of the medial recti, hypertropia increased 7 Δ in 23% and decreased 7 Δ in 34%. After recessions of the lateral recti, the hypertropia increased 8.3 Δ in 36% and decreased 7.8 Δ in 23%. They conclude that postoperative hypertropia depends on many factors and the degree is unpredictable. Displacement, up or down, of the muscle operated on is not the explanation. They operate on the vertical after the horizontal except where the deviation is large.

Alternating Hyperphoria.—Anderson [108] has given us another of his thoughtful, detailed surveys, this time on latent nystagmus and alternating hyperphoria. Out of 34,000 patients, there were 138 cases of congenital nystagmus, and 20 of these were of latent nystagmus, of which 13 were considered typical. All these patients had alternating hyperphoria. Latent nystagmus becomes manifest on occlusion of one eye and usually on conjugate deviation, when the slow component is away from the side looked at. All but 1 patient had strabismus: 11, esotropia, 5, exotropia; 11, overaction of both inferior obliques. Alternating hyperphoria he divides into three groups: (1) alternating sursumduction—upward deviation of either eye when binocular vision is interfered with and there is no muscle imbalance; (2) alternating hyperphoria—similar upward deviation associated with even the least muscle imbalance; overaction of the inferior obliques; (3) alternating hypertropia—upward deviation of either eye, obvious in binocular vision on alternate fixation, due to paresis of the depressors. He questions whether (1) and (2) are different and wonders if overaction of the inferior obliques can be classed as a sign of past or present paresis. He concludes that latent nystagmus may be regarded as an entity. The exciting agent appears to be asymmetrical stimulation of distance receptors. Alternating hyperphoria is always associated with signs of imbalance of the obliques and sometimes the recti. It may be due to arrested development of the oculomotor mechanism. Difficulties at birth are common and may cause both. Latent nystagmus appears to decrease with age. As always, one is impressed with Anderson's thorough review of the literature, and while his conclusions may not solve many of our problems, they may stimulate our thinking along the proper channels.

In a new article, Crone [109] has gone over much of the same material covered in his monograph on alternating hyperphoria, last year. The syndrome consists of (1) squint from birth (50%); (2) hypertropia in lateral gaze, the nonfixing eye being turned up in most; (3) pendular and rotatory nystagmus, which with occlusion is changed to jerk nystagmus with the rapid phase toward the fixing eye; (4) alternating hyperphoria, the covered eye rotating up slowly; (5) excycloduction of the covered eye and incycloduction of the fixing eye, and (6) torticollis toward the fixing eye. He now

divides his 113 cases into five groups: (1) esotropia surso-adductoria, with hypertropia of the squinting eye in adduction; (2) esotropia surso-abductoria, with hypertropia of the squinting eye in abduction; (3) alternating hyperphoria without horizontal deviation; (4) exotropia surso-adductoria; (5) exotropia surso-abductoria. The esotropias are generally in Group 1, and the exotropias are almost always in Group 5. He discusses the question of whether the innervational disturbance of alternating hyperphoria is a disturbance in congenital binocular innervation or in monocular innervation. He finds indications that there may be a disturbance of the monocular optomotor reflexes. He assumes that optical stimulation of one eye causes asymmetrical increase in tonus of the eye muscles. He repeats the explanation that he gave previously that these cases are caused by deficient motor impulses from the lower nasal quadrants of the retinas.

In spite of the noble efforts of these authors, I still find this subject very confusing. Apparently Crone uses "alternating hyperphoria" as synonymous with "dissociated hyperphoria," and both he and Anderson mention "occlusion hypertropia" without differentiation. Just what relation upshoot in adduction has to this group is not clear, although Crone says that there is a definite relationship and found them frequently associated. Anderson's third group apparently includes both. Pointing out the frequent association of latent nystagmus and alternating hyperphoria is of interest, but it does not simplify the problem. It is obvious that many more of these cases will have to be studied before we shall understand them fully.

SUMMARY

While the number of articles appearing in 1954 was somewhat larger than last year, there were no outstanding literary events to mark the year. One can still find support in the literature for almost any belief that one may hold, but certain trends seem to be indicated. In an unprecedentedly short time, Bangerter has become a major influence in the treatment of amblyopia. While most authors are mentioning his work, we will have to wait to find out how much his methods are actually adopted. There has been a striking increase in the number of articles on anomalous correspondence and orthoptic training from central Europe, indicating, I believe, a very definite change of attitude. Several authors from this same area have divided esotropia into two distinct groups: monocular esotropia with amblyopia, and alternating esotropia with anomalous correspondence. This rather arbitrary classification would seem to have therapeutic implications that might be dangerous. An increased interest in the electrical recording of eye movements is indicated, but as yet no very impressive results have been reported. The trend toward symmetrical surgery for comitant strabismus, which began last year, is gaining momentum.

CONCLUSIONS

After writing this review for three years, certain impressions about the literature on strabismus should be recorded. It is just about impossible to report the results of the treatment of a series of cases so that it will mean much to anyone else. The reason for this is the practical impossibility of giving enough definitions and criteria, particularly as to the tests used and their interpretations, to let a reader make a true evaluation of what was done. It may be that if we continue to strive for precise definitions, uniform terminology, standard methods of examination, and agreement in their interpretation, the day may come when this will not be true. My over-all impression is that definite advances are being made in the understanding of the physiology of strabismus and in the application of this knowledge to its treatment.

30 W. 59th St. (19).

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Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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Dec. 16, 1954

Leiomyoma of the Iris: Report of a Case and Review of the Literature. LT. WAYNE L. ERDBRINK (MC), U. S. N.

A case was presented of leiomyoma of the iris in a 20-year-old white man located in the inferior nasal quadrant of the left eye. The vision, intraocular tension, and chamber angle in the involved eye were normal. The clinical diagnosis was leucosarcoma of the iris. P^{32}-uptake studies were negative, indicating nonmalignancy of the tumor. The tumor was removed with a basal iridectomy, and the patient has been followed for 10 months, without evidence of recurrence. Pathologic examination of the tumor met the criteria for leiomyoma of the iris.

The world literature was surveyed, and 19 case reports were found. The historical and clinical characteristics of these cases and of the present case were summarized.

Conservative surgical management of any case of a localized tumor of the iris was stressed.

DISCUSSION

DR. JOHN MCGAVIC: I had the opportunity of examining the patient clinically when he came to the Wills Eye Hospital for photographs and P^{32} studies. As we go on with this discussion, I think you will agree with me that the ophthalmic pathologist would welcome a divining rod, like P^{32}, as much as the clinician, for the microscopic diagnosis is sometimes almost as difficult as the clinical diagnosis. This patient's tumor fungated into the anterior chamber more than does the usual leiomyoma and did not produce ectropion of the uvea, as is the rule. The literature says that ectropion uveae is present in about 35% of cases, but I think it is present much more frequently than this.

(Slide) This slide shows a leiomyoma of the iris which is near the pupillary border. This type is thought to arise from the sphincter rather than from the dilator. This area represents a denser nuclear arrangement than the rest of the tumor. Associated with this there is a large cyst, which lies between the two layers of epithelium on the posterior surface of the iris. In this instance, it extends into the pupillary area and was seen as a large dark mass. Some leiomyomas may be associated with dark masses, while they themselves are gray to yellow. This type of cyst is not uncommon in the published pictures of leiomyoma.

The first patient reported was a man about 40 years of age. The next one was the second case that the late Dr. Albert Frost had found. He made the diagnosis clinically. Incidentally, his patient had glaucoma. I think we have had 3 cases with glaucoma that I know about, as opposed to the 1 out of 20 cases reported by the essayist.

(Slide) This slide shows a leiomyoma stained with the trichome method, of the type that extends along the surface of the iris and tends to produce ectropion uveae. There is usually more ectropion than in this case. This patient was 28 years of age and had secondary glaucoma. (Slide) This is from the case described by Moulton and Moulton, that of a man, aged 56, with an enlarging growth noted for a year and a half. It measured 5 by 7 mm., filled the anterior chamber, and pressed against the cornea. This case was mentioned by Lieutenant Erdbrink.

(Slide) The next slide shows a low-power view of the globe with a leiomyoma involving the base of the iris and filling the angle, a situation which is said in the

literature not to occur very often. We have two instances of this. Secondary glaucoma is evidenced by the cupping of the nerve head.

The next case is one of those in which the glaucoma was rather acute. The cornea was steamy, and vision was reduced to counting fingers at a few feet. The diagnosis of melanoma was made by three persons, and that of leiomyoma by three others. (Slide) This slide shows why the term fibroid is applicable to leiomyomas. The amount of spindle-shaped fibrous cells here is quite marked, and, as you look at the microscopic slides, you will see long, spindle-shaped cells which are quite different from these; the two parts may be present in the same tumor. (Slide) Next is a slide from the same tumor showing the spindle cells under higher magnification; the other part of the tumor showed the fibrous portion.

(Slide) This specimen I have not seen before. The slide was made by someone else, and I picked it up from the essayist as we came in. You can see that the cells are quite variable in their appearance. This is due in part to their being cut in different planes. These cells are more or less spindle-shaped. Some appear round or oval, but they are probably cut largely in cross section. The next slide is a lower magnification, showing that some of the cells are really quite spindle-shaped. When you get a cell with malignant melanoma on the side, you will find it is less spindle-shaped than the term spindle-cell melanoma implies.

(Slide) This is from a biopsy specimen in which all the iris from here forward is involved by a tumor. There is a different type of cellular arrangement on the surface of the iris. The cells tend to spread along the surface of the iris, as epithelium is inclined to do.

(Slide) I think this is the best picture of all in that it shows the tendency of nuclei to palisade and the spindle-cell formation. Many of the cells appear spindle-shaped, more so under the microscope, but they grow in such different planes that it is hard to photograph them and show the spindle-cell arrangement properly.

(Slide) The next specimen is shown through the courtesy of Dr. James Shipman. The patient had a tumor of the lower portion of the iris which proved to be a nevus, as you can see from the nests of cells that are present here. In some of the published cases of leiomyoma, the photographs give the impression that the tumor may very well be of the nevus-cell type, and this would explain the difference in behavior of some of these cases. This is a higher-power section to show the arrangement of the nevus cells. You can see that they are more nearly rounded and oval, rather than spindle-shaped, and that they are also arranged in nests.

(Slide) This is a melanoma of the iris, which we felt was malignant. On section you notice it appears like a leiomyoma. The cellular detail of melanoma can vary from epithelioid to a spindle-cell type. This is a malignant melanoma of the iris involving also the ciliary body, and it is nonpigmented. On section you could not tell it from a leiomyoma, except under higher power.

Melanomas of the iris are malignant in only about 9% or 10% of cases. Leiomyomas are only theoretically malignant. No proved case of metastasis from an iris tumor has been reported. Only one leiomyoma of the ciliary body has been reported, and this did not metastasize. We must agree with the essayist that removal of iris tumors by simple basal iridectomy is certainly the treatment of choice.

I would like to congratulate the essayist on the splendid presentation of his paper. I have nothing to add to or change in what he said.

DR. I. J. EISENBERG: We have now been using P^{32} almost three years. During this period we have tested 10 to 12 iris lesions by our technique. In this series there were two cases of malignant melanoma involving the iris and ciliary body, proved by histologic sections, which showed a positive uptake of P^{32}. We have also verified two cases of nonmalignant lesions of the iris, which gave a negative P^{32} concentration. We also have four cases under observation. Everyone who has examined these lesions feels that they are so-called malignant melanomas, three of them showing all the classic signs, encroachment of the angle, pigmented cells in the angle, and apparent increase in size, one case even showing destruction of iris stroma. In these four cases, the P^{32} test was negative. A possible explana-

tion is that even though these lesions show signs of malignancy by the established criteria, they may not be true malignant lesions in the sense that metastasis will occur, and the metabolic rate may be no different than the surrounding normal iris stroma. This low metabolic rate and lack of clinical evidence of metastasis are further borne out by Reese's analysis of 32 proved cases of malignant melanoma of the iris, only 3 of which showed evidence of metastasis.

Our results and Reese's report raise a pertinent question in my mind as to whether lesions of the iris not involving the ciliary body should be considered clinically malignant, even if the pathologist reports a malignant melanoma. Only time and more facts will resolve this question. Nevertheless, from the information already known, I should like to urge a "go-slow" attitude toward enucleation of an eye with a diagnosis of malignant melanoma of the iris.

Congenital Bilateral Abducens: Facial Paralysis (Möbius Syndrome). DR. JULES M. YASUNA and DR. NATHAN S. SCHLEZINGER.

The literature was briefly reviewed, and two cases of this syndrome in children were described. Both children had congenital bilateral facial and abducens nerve paralyses with retention of ocular convergence, but little or no internal rotation of the eyes upon attempted lateral conjugate gaze. Associated anomalies consisted of a clubfoot in one boy and a digital deformity of the hand in the other boy. Noteworthy in the comprehensive diagnostic survey of these two cases were the absence of nystagmus and extraocular movements in response to caloric vestibular stimulation, the normal creatine and creatinine urinary assays, and the markedly reduced quantitative response observed in the electrical reactions of the facial muscles. Exploratory operation in both cases revealed grossly normal lateral rectus muscles with free movement of the eyes in response to forced duction maneuvers. The muscle biopsy was reported as showing some abnormality in the form of loss of striation and peripheral migration of the nuclei in some of the fibers, but these changes were not conspicuous and were considered as not consistent with dystrophy but as probably consistent with a relatively slight degree of muscle atrophy. On the basis of the data obtained in the study of these two children, it is postulated that the Möbius syndrome is the result of a congenital brain stem lesion which is predominantly supranuclear in its effect upon the abducens nerves and either nuclear or infranuclear in its effect upon the facial nerves.

DISCUSSION

DR. HOWARD BEDROSSIAN: Henderson, in 1939, reported 61 cases of the Möbius syndrome, in 45 of which abducens palsy was present. Of those 45 patients, 23 showed a convergent squint. The other 22 had straight eyes. In reviewing the literature concerning this syndrome, I found that one of the most interesting features concerns the pathology that is postulated. With regard to the pathology, two schools of thought seem to exist: one, that which believes that a primary aplasia of the muscle exists, due to a mesodermic defect, and the second, that which believes there is a nuclear or ectodermal defect.

Evidence of muscle defects has been shown by Shenkle, Obersteiner, McDonald and Mittendorf and, finally, Heuck. The outstanding report is that by Heuck, in 1897. He reported a family in which there was a mother and three children, all of whom had eye muscle defects, including bilateral ptosis, inability of outward movement of either eye, and inability to raise or lower the eyes. One of the children died at age 18, and autopsy showed that all the rectus muscles except the medial were inserted in an abnormally posterior position, some of them as much as 2.5 mm., behind the normal, and that nearly all the muscles were too short (as much as 10 to 11 mm. short in the case of the inferior rectus muscle). He concluded that the faulty insertions and incomplete development adequately explained the defective motility. The nerves were not examined.

According to the other school of thought there is a nuclear or ectodermal defect. Two main types of nuclear involvement are suggested. One type is that of aplasia of the nuclei. Heubner's case, reported in 1900, which is cited in Duke-Elder's and Walsh's textbooks, is an example of this type. The other type of nuclear

involvement is that of degeneration, as shown in a case reported by Rainy and Fowler in 1903. In this case the patient had facial diplegia but no ocular involvement, and pathologic study showed degenerated nuclei and nerves, and atrophied muscles. Other authors, such as Spatz and Ullrich, and Balint, have also reported nuclear pathology.

The possibility that two types of pathology may exist is well summarized in an interesting article by Leszynsky, in 1897. He stated that in the few cases examined post mortem there has been found complete absence of the muscles of abnormal insertions. "Neuropathology teaches that fibrous degeneration and complete absence of a muscle may be due to a primary nuclear atrophy, while, on the other hand, the nuclear atrophy may result from a loss of function or atrophy of muscle."

I would like to add to Dr. Schlezinger's three cases a case of muscular pathology.

Mr. N. D., age 35, complained of inability to gaze laterally since birth. The previous medical history was that of left herniorrhaphy and spinal arthrodesis at the ages of 16 and 18, respectively. Visual acuity was correctible to 6/6 in each eye. Ocular movements showed ability to elevate and depress the eyes normally. He was unable to abduct or dduct either eye. Convergence was present, with normal pupillary reactions. His near point of convergence was 20 cm. from the nose. Bell's phenomenon was present. The patient showed first-, second-, and third-degree fusion in eyes front. His prism divergence was 10/5 and his convergence 20/10. Optokinetic nystagmus on the rotating drum test showed no response to either side. The general finding was that of a slightly masked facies, but there was no definite facial paralysis. He was able to smile, but not quite as normally as most persons. He had diminished hearing on the right and hammer toe on the right, and a cousin had webbed fingers.

A Bárány test performed at another institution suggested a midline infratentorial lesion. In view of this finding, Bárány and caloric tests were repeated at the Graduate Hospital. The report of the consultant was as follows: "Hypoactive reflexes to both mass caloric and Bárány testing. Bárány testing is of decreased value because of lack of nystagmus. However, if the patient's eye complaints are on the basis of intracranial pathology, such pathology would be located in the midline to coincide with decreased response to vestibular stimulation." Operation was performed, but before surgery, while the patient was under anesthesia, a forced duction test was done. We were not able to abduct or adduct either eye at all, but we could easily elevate and depress the eyes with the forceps. The medial and lateral rectus muscles were then explored and found to have many check ligaments. Tenon's capsule in the region of the muscle insertions was about as thick as the muscle itself. The lateral rectus muscle was inserted 12 to 13 mm. from the limbus. The medial rectus muscles were also inserted slightly more posteriorly than normal. An attempt was made to stimulate directly with the electric current while the patient was under anesthesia. Both alternating and direct currents were used in the usual manner, but no response was obtained. The technician performing the electrical stimulation felt that no conclusions could be drawn because the patient was under anesthesia. Postoperatively, no improvement whatever was obtained in abduction or adduction, although the muscles were freed without being detached.

In summary, I would like to make the following comments:

1. In view of the questionable facial diplegia, this case may not be a typical example of Möbius syndrome; nevertheless, it could be classified with that group of congenital anomalies.

2. The pathology in this case is definitely peripheral, in the muscles.

3. It is hoped that in the future more cases of this type will be recognized and that forced duction tests and exploration of the muscles will be performed for diagnosis. If this is done, future correlation of muscular aplasia with nuclear aplasia may be obtained.

4. A knowledge of existent congenital peripheral ocular muscle pathology will help to prevent the misinterpretation of vestibular tests in suspected brain tumor cases.

DR. FRANCIS HEED ADLER: The question whether the pathology in this case is a muscular dystrophy or a nerve tract lesion is of real importance. Since these patients retain convergence, and yet have no adduction of either eye on versions, it is likely that the lesion is in the posterior longitudinal bundle. One might argue, however, that the failure of the right eye to adduct in levoversion, and of the left eye to adduct in dextroversion, is merely due to lack of training, since a child with a congenital bilateral sixth nerve palsy will have no occasion to use the medial rectus of the opposite eye in versions. This argument is plausible, since we see a similar failure under other circumstances. In children with congenital ptosis, for example, one frequently finds failure of the eyes to move upward on command after the lids have been manually elevated, merely because the child has never used upward gaze. After some practice and urging, the eyes will turn up, demonstrating that the muscles for sursumversion are not paralyzed. In certain forms of strabismus, the habitually squinting eye may not abduct, giving rise to the appearance of a sixth nerve palsy, but after the fixing eye has been patched for a week or so, abduction is regained. This, however, cannot be the case here, since we find that the medial recti do not work in versions, no matter what the stimulus. It is well known that these patients do not show any horizontal optokinetic nystagmus, or any horizontal nystagmus as the result of vestibular stimulation—i. e., the Bárány chair test or douching the ear. In spite of this, they retain good convergence, proving to me that this syndrome must be due to lack of development of nerve tracts involving the sixth and seventh nuclei and the posterior longitudinal bundle.

DR. I. J. EISENBERG: Dr. Adler, in sixth nerve palsy are there any cases in which you do not get estropia?

DR. FRANCIS HEED ADLER: Oh, yes, I think in only about half of them. Dr. Bedrossian quotes Henderson's paper. About half the cases have esotropia, as I recall, and in the others the eyes were straight in the primary position.

DR. NATHAN S. SCHLEZINGER: I am wondering how a muscular disorder postulated in the case mentioned by Dr. Bedrossian could explain cases in which facial involvement was also present. In other words, we cannot consider facial paralysis a consequence of some abnormal insertion of the muscle, but, rather, regard it as the result either of a myopathy or of a nuclear displasia.

Blepharophimosis. DR. EDMUND B. SPAETH.

The author reviewed the history of the rather difficult ophthalmic plastic surgery concerned with this defect. He also discussed the basic anatomic pathology to illustrate the essential requirements for the correction of the defect.

The epicanthal fold, when this lies largely in the lower lid, is the most difficult to correct. The author presented a new operation for the correction of the so-called epicanthus inversus—a term which he thinks rather superfluous. The presentation was based upon the satisfactory correction of over 70 cases of this deformity, and the operation has no great technical difficulties. He plans to present the entire paper shortly for publication.

George F. J. Kelly, M.D., *Chairman pro tem*
William E. Krewson 3rd, M.D., *Clerk*
Jan. 20, 1955

Hydrops Corneae from Keratoconus: Case Presentation. DR. JOSE FIOL-BIGAS.

The case presented is one of that very rare complication of keratoconus resulting from the rupture of the posterior surface of the cornea, involving the endothelium, Descemet's membrane, and the corneal lamellae themselves.

Recently, Dr. Townley Paton discussed a case he had had of this same complication which he handled in a manner different from ours. The results obtained by the procedure to be mentioned were most satisfactory.

The patient was a 22-year-old man who had keratoconus, diagnosed at the age of 12. Upon admission, keratoconus on the left, rather extensive, but without any serious or important complicating factors, was noted. The right eye showed a mildly irritable eye with tremendous edema of the entire cornea. The thickness

of the cornea at the limbus measured, as accurately as is possible under the slit lamp, over ¼ in. This edematous cornea thinned very rapidly toward the site of the former keratoconus apex, slightly below and nasal to the center of the cornea, where the edema decreased to a thickness of about ⅛ in. In this way the corneal thickness reminded one of a cross section of an anterior convex and a posterior highly concave lens of meniscus type. The cornea was a faint grayish blue and was barely translucent. One could not see the texture of the iris through its substance. Visual acuity was light perception and projection.

The keratoconus, in this varying bilateral state, had been present for several years unchanged and had apparently been stationary. The patient was using contact lenses, and had been using them for some time, with very satisfactory visual acuity, according to the history obtained from him. The complication in the right eye present upon first examination had been developing for the past month to six weeks. It had become most severe rather suddenly and abruptly in the 10 days immediately prior to his visit to the office.

The patient was admitted to the hospital, and shortly thereafter, with the use of local anesthesia, a ring of eight deep diathermy punctures was made into the corneal stroma, but not through it. A Walker pin 1.5 mm. in length was used for this. It was remarkable to see almost immediate beginning of corneal clearing with the drainage of the fluid, presumably aqueous, which had infiltrated into the corneal stroma. The eye was atropinized, and a pressure dressing was applied to the right eye and a binocular bandage to both eyes.

Forty-eight hours later the dressing was removed in the operating room, with the patient under general anesthesia, and a 7 mm. circular corneal graft was implanted with direct approximation sutures. At this time the corneal stroma was normal in appearance, in thickness, and in texture. There was no problem whatsoever connected with the corneal texture insofar as the introduction of the sutures was concerned. A normal resistance to the passage of the needle and a normal resistance of corneal structure as the sutures were tied were noticed. Postoperative convalescence was uneventful, and the patient's visual acuity at the present time is 6/6—4, with a +1.50 sph ⌒ —11.00 cyl, ax 172½ correction.

The corneal grafting has since been done in the left eye as well. Surgery on this side is so recent that the patient does not as yet have a correcting lens in that eye. Visual acuity at his last examination, however, several days ago, was 6/6—3 with a —11.50 cyl, ax 175.

As stated, the purpose of this presentation is to describe the result obtained with an eye which at first examination seemed to be in an almost hopeless condition, and the rapid favorable response from a relatively minor surgical procedure carried out prior to the grafting.

A New Apparatus for Visual Screening in Schools. Dr. Samuel M. Diskan (by invitation), Atlantic City, N. J.

The recent organized assault against inadequate visual screening programs in schools urgently demands that the ophthalmologist propose an answer to this ever-growing problem.

It has become increasingly obvious that tests for visual acuity alone cannot constitute an acceptable visual screening program.

The Atlantic City (Diskan) Eye Test was developed to detect at 20 ft. (1) impaired visual acuity, (2) excessive manifest hyperopia, and (3) abnormal eye muscle balance.

The eye-muscle screening test is original and employs red and green light to suspend fusion and dissociate the visual functions of the two eyes. It measures lateral and vertical phorias simultaneously and is an almost instantaneous method of separating the abnormal from the normal eye muscle balance.

A preliminary study of 799 Atlantic City school children with this test revealed that 18% had impaired visual acuity, 2.4% excessive manifest hyperopia, and 1.4% abnormal eye muscle balance. The average testing time per child was 50 seconds.

The test was fast, effective, accurate, and easily understood and did not give excessive false referrals. The apparatus is inexpensive and easily portable.

The author feels that the Atlantic City Eye Test is an improvement over existing visual screening tests and that it may be the answer to the agitation for an increased scope of visual screening in schools.

Oculomotor Manifestations of Certain Lesions in the Brain Stem. Dr. Thomas R. Hedges Jr. (by invitation) and Dr. Francis Heed Adler.

Three cases were presented in which lesions of the brain stem produced unusual oculomotor manifestations. The first was a case of unilateral internuclear ophthalmoplegia in a child in whom ventriculography showed a lesion in the region of the pons, causing a defect in the aqueduct and fourth ventricle. No autopsy was obtained, but the clinical course and neurologic findings suggested that the etiologic agent was an expanding lesion, possibly a pontine glioma. The second case showed bilateral internuclear paralysis, which progressed to complete loss of lateral conjugate gaze. Vertical movements and convergence were retained. The rapid regression of these findings suggested that the etiology in this case was disseminated sclerosis. The third case was one of complete loss of vertical movements initially with a transient left hemiparesis. The patient recovered downward movements and upward following movement, but command movements in upward gaze remained absent for two and one-half years. A lesion, presumably vascular, in the region of the superior colliculus probably caused these motor defects, classified as Parinaud's syndrome.

(Illustrative moving pictures were presented in each case.)

Dr. George P. Meyer, Camden, N. J.: I would like to be enlightened about the innate transient nature of demyelinating diseases. I was under the impression demyelination was an irrevocable process. I think Dr. Hedges said that this lesion might have been of demyelinating nature, since this man recovered.

Dr. Francis Heed Adler: One of the characteristics of the demyelinating diseases is the rapidity with which they come on and with which they change their character. Witness the scotoma in multiple sclerosis, which is characteristically very sudden in onset, involving complete cessation of transmission of impulses, so that there is an absolute scotoma, which in the course of a relatively very short period clears up entirely. The moment you get a picture of rapidity of change in a deep-seated disturbance of function, you say to yourself that it is probably a demyelinating disease. The same thing is true also of the motor side. Those deficits in ocular motility of sudden onset and great variability that clear up rapidly point to the demyelinating group of diseases—not specifically multiple sclerosis, but DeVic's disease, encephalomyelitis diffusa, Schilder's disease, and the other forms of demyelinating disease. Dr. Hedges and I wish to emphasize that there is great specificity of these syndromes in localization. This area is a tiny one of some centimeters, running from the third to the sixth nerve nucleus, and you can pinpoint lesions by these phenomena very well. Although we have great specificity in localization in the central nervous system, we would like to point out that our cases, and those in the literature, demonstrate that the same signs can be caused by any kind of pathology. The syndrome may be a duct tumor, vascular disease, or demyelinating disease.

Dr. E. Howard Bedrossian: The authors are to be congratulated on their very informative paper. It is interesting to compare their second case with the cases of the Möbius syndrome that were presented at last month's meeting of the College of Physicians. In the case presented this evening definite acquired neurologic disorder of the brain stem gave exactly the same findings of bilateral abduction and adduction paralysis (with the exception of the seventh nerve paralysis) as were present in the Möbius syndrome. The Möbius syndrome, of course, is congenital and is thought to be due to nuclear aplasia in some cases.

Last month I presented a case which had congenital bilateral paralysis of abduction and adduction due to peripheral changes in the muscles. This case also had the same clinical findings as Dr. Hedges' Case 2. Therefore it is important to keep in mind the fact that these three pathologic conditions may resemble each other clinically.

The history would be very significant in differentiating congenital from acquired paralysis of abduction and adduction. The forced duction tests would be very useful in differentiating peripheral muscle defects from a nuclear lesion in the congenital cases.

Coats's Disease: A Clinical and Pathologic Study. Dr. William C. Frayer (by invitation) and Dr. Harold G. Scheie.

The authors presented clinical and pathologic reports on two eyes from children which showed complete retrolental membranes and were enucleated because of a suspicion of tumor. Histologic evidence of massive subretinal hemorrhage with partial organization was found in both eyes. They were considered by the authors to be examples of the symptom complex known as Coats's disease. In one of the eyes no definite etiologic agent could be discovered to account for the subretinal hemorrhages. In the other eye hemorrhages were thought to be associated with retinal changes secondary to subdural hematoma.

The differential diagnosis of pseudoglioma was reviewed. The current concept of Coats's disease was also discussed.

DISCUSSION

Dr. William McCarty: It would be interesting to know whether P^{32} studies were done.

Dr. William C. Frayer: We did not do P^{32} studies. We felt that even a positive result would not have altered our decision to enucleate these eyes.

Edmund B. Spaeth, M.D., *Chairman*

William E. Krewson 3rd, M.D., *Clerk*

March 17, 1955

Diagnosis and Treatment of Interesting Lesions of the Limbus. Dr. Charles E. Iliff, Baltimore.

A discussion of interesting lesions of the limbus embodied some tumors, some inflammatory lesions, and some degenerative changes which had offered a problem either in diagnosis or in treatment. Emphasis was placed on the clinical picture. Although the pathologic findings were not taken up in detail, it is in them that the final weight of diagnosis lies, and the importance of a biopsy specimen cannot be overemphasized.

Differential diagnosis of limbal tumors is greatly aided by a knowledge of the age of the patient and the duration of the lesion. In children, benign tumors of congenital origin are most commonly noticed very early in childhood, though some escape detection until puberty, or even later. In adults, there is a very sharp increase in the frequency of malignant tumors. Thus, for practical purposes, we can consider most limbal tumors in children as benign, and in such cases therapy may be conservative. In adults, on the other hand, in whom a high percentage of the tumors are malignant, therapy must be more radical, making complete removal of the malignant growth of primary importance.

The dermoid, angioma, nevus, melanoma, papilloma, and epithelioma offer especial points of interest, either in diagnosis or in treatment. The method using iodine for the removal of epithelial tumors which have not penetrated Bowman's membrane was described in detail.

The treatment of some inflammatory limbal lesions, of pterygia, and of Mooren's ulcer was discussed. The success of the partial penetrating graft has altered our view of the prognosis of corneal ulcers. The possible use of frozen cornea is suggested when fresh material is not available. The work being done in this field was briefly mentioned, and slides were shown of a successful grafting of frozen material.

DISCUSSION

DR. JOHN FINLEY: I remember a large dermoid cyst in a case of Dr. Iliff's which involved the complete temporal region of the palpebral conjunctiva. I would like to ask the method of incision; I have had two like that.

DR. CHARLES E. ILIFF, Baltimore: I removed the cyst by a lamellar keratectomy, going down to the clear cornea and then excising the whole lesion, and sliding the conjunctiva laterally as much as I could. In this case I also took conjunctiva from the other eye to cover the defect.

DR. JAMES S. SHIPMAN, Camden, N. J.: I would like to ask Dr. Iliff what technique he uses for removal of ordinary pterygium and what particular knife.

DR. CHARLES E. ILIFF, Baltimore: I like the Gill knife. I think it gives the best splitting procedure that we can get. The Gill knife is slightly cupped and has a rounded, but very sharp, cutting surface, and one can split the cornea. I use a Gill knife to outline the pterygium and remove it in toto, whether it is small or large. I do not treat all pterygia by irradiation. I do not think it is necessary to use cortisone postoperatively for the small ones, but if I notice any vascular signs of inflammation, I irradiate the area.

DR. I. J. EISENBERG: You mentioned radiation. It is always beta or gamma rays?

DR. CHARLES E. ILIFF, Baltimore: I use beta radiation from the Burman applicator, which contains radon. It has a certain percentage of gamma rays, but the output of gamma radiation is low as compared with that of the beta rays.

DR. HAROLD G. SCHEIE: Dr. Iliff, what changes have occurred in some of your patients who have had fairly large amounts of beta radiation? For example, I saw several patients of yours when I used to go to Valley Forge Hospital in 1947 and 1948. Have any of those patients been followed, and, if they have, what do they show today? I have never seen any of them.

DR. CHARLES E. ILIFF, Baltimore: Some of them have shown dilated vessels behind the squamous cells, such as occur with irradiation elsewhere. I think these patients had too much treatment. I have not seen any breakdown of tissue in our group, but there are reports of a certain amount of hyperplasia. The lesion is not as bad as I showed here, but there is a sloughing of superficial cells, such as you see with a general reaction of the eyes. One patient lost all her lashes. One of the early cases, she was overtreated, and she had a scaling of her lids. Whether this was due to the beta radiation or to other factors as well I cannot say; however, the thing that we have noticed in the patients whom we have overtreated has been the matrix of the dilated vessels. You can tell a case that has been heavily treated.

JOINT MEETING OF NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY WITH NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Jan. 17, 1955

SYMPOSIUM ON RETINAL DETACHMENT

Moderator: John M. McLean, M.D., *New York*

Members of Panel: Graham Clark, M.D., *New York*

Charles L. Schepens, M.D., *Boston*

Donald M. Shafer, M.D., *New York*

James S. Shipman, M.D., *Philadelphia*

DR. JOHN M. MCLEAN: Dr. Clark, in which cases would you use a scleral resection as a primary procedure? Why are scleral shortening operations better than classical diathermy in these cases?

DR. GRAHAM CLARK: For the purposes of this discussion, all detachments may be divided into three categories:

1. "Simple detachments," or those which will reposition themselves on removal of the subretinal fluid.

2. "Complicated detachments," or those in which mechanical forces, such as retinal shrinkage, surface membranes, persistent strong vitreous traction, or agglutinated retinal folds, prevent the retina from repositioning itself against the wall of the eye.

3. "Borderline," or "doubtful," detachments, or those in which the surgeon does not have sufficient evidence to put them in Class 1 or 2 or those with the retina under weak vitreous traction, which, while allowing the retina to get back to the wall, will, by the persistence of its pull, redetach it.

In Classes 2 and 3 scleral shortening is the procedure of choice and, when indicated, should be the primary procedure, for there is ample evidence that the diathermy reaction causes retinal and vitreous shrinkage and not only may convert a simple detachment into a complicated one but may put a complicated one beyond the reach of even the scleral shortening operation. In addition, it causes scleral necrosis and makes the scleral shortening procedure difficult or impossible in that area for two or three months.

In these last two groups lie the detachments in aphakia and high myopia and those of long standing, as well as the detachments characterized by foamy equatorial ridges and multiple holes and those with angular holes whose edges are stiffly everted; but these surface characteristics are not sufficient criteria in themselves.

The decision must rest on the most complete knowledge obtainable of the mechanical factors involved. A period of repeated observations with the patient resting comfortably in bed with the eyes bandaged for 48 hours should be a minimum.

Dr. Charles L. Schepens, Boston: Dr. Clark tells us that cases which belong to Class 1 should be treated by simple diathermy. However, it is my experience that there are instances in which a detachment appears favorable before operation but reacts very poorly to a correctly applied diathermy treatment. I am often unable to classify a case until after the operation, and that is of limited practical usefulness. Consequently, a choice of operative procedures which is based on the appearance of the detachment before operation must lead to many disappointments. The only logical course therefore is to treat every case as if it were unfavorable.

As far as the classification itself is concerned, I agree that the presence of aphakia or high myopia may be important; however, in my opinion, the existence of fixed retinal folds is of such overwhelming importance that this should be the deciding factor in classification. There are patients with aphakia or high myopia who do extremely well, but all patients tend to do poorly once they have fixed retinal folds.

I strongly agree with the statement that extensive diathermy greatly diminishes the chances of reattachment. Therefore it is extremely important to apply the treatment as gently as possible.

Dr. Donald M. Shafer: I cannot see why any procedure that is liable to convert a so-called Class 1 case into a Class 2 or Class 3 case should be utilized at the outset if there is available some procedure which is less likely to end in a Class 3 case. I feel that a less destructive or involved primary procedure might save a certain number of cases from being converted from Class 1 to Class 2 or 3.

Dr. James S. Shipman, Philadelphia: I must say I am not overwhelmed by the fear of too much diathermy. To my knowledge, I have seen only a single eye that was destroyed by using too much diathermy, and that was many years ago. Personally, I feel that diathermy is still the major weapon to use against retinal detachment; and if I had my choice of diathermy or resection, I should take diathermy. I still rely chiefly on this, even though I do resection. I do just as much diathermy as though I did not do a resection. I might not use as strong a current as I would otherwise, but first of all, as I shall try to show with a few slides, I put in my Walker pins for my real insurance in holding the retina. The criteria I use in deciding when to do a resection and when not to do it are those expressed by other members of the panel, and I agree that, whatever the reason may be, retinal folds or scarring, if the retina does not go back into place within

five days—and I give the patient five days in bed in a dependent position—I then do a resection. I think one must allow more than 48 hours for this to occur. That is the criterion I go by. I appreciate Dr. Clark's comments, but I must differ, as indicated.

DR. JOHN M. MCLEAN: Dr. Schepens, what are the advantages of scleral buckling with a plastic implant over classical diathermy or scleral resection?

DR. CHARLES L. SCHEPENS, Boston: A scleral buckling operation generally consists of the following steps: 1. The sclera is adequately exposed. 2. Localization of all the retinal breaks is made by ophthalmoscopy, a mark being made over each break on the sclera with a flat diathermy electrode. 3. A scleral lamella 4 to 6 mm. in width and one to three quadrants in length is excised. It is essential that the middle of the excised strip of sclera should be slightly posterior to the retinal breaks. However, when the breaks are anterior to the equator, the middle of the excised strip should be placed at the equator. 4. The area of scleral excision is covered with surface diathermy. 5. Mattress sutures are placed on the edges of the area of excision. 6. Subretinal fluid is released through a perforation made in the area of scleral excision. 7. A tube of polyethylene, 1.00 to 1.50 mm. in diameter, is placed under the sutures. 8. The sutures are drawn and tied over the polyethylene tubing. 9. A connection is made with surface diathermy between each end of the buckle and the ora serrata.

The advantages of the method may be summarized as follows: The treated area is pushed toward the retina as much as evacuation of the subretinal fluid will permit; this insures closure of the retinal breaks at the time of surgery, instead of later. It is unnecessary to give the patient preoperative bed rest, as this might diminish the quantity of subretinal fluid available and would make the buckling operation difficult and less effective. Postoperatively, the period of bed rest is limited to four to eight days; after this pinhole glasses are not used. It is remarkable that if the same preoperative and postoperative schedule is followed in cases of simple retinopexy, failure will often result.

The most important advantage of scleral buckling is that it is a very effective means of combating fixed retinal folds. The fixed fold is the major obstacle to reattachment of the retina. The frequent occurrence of an equatorial fixed fold makes it necessary to place the buckling no farther anterior than the equator. Because of its effectiveness against fixed retinal folds, scleral buckling makes it possible to reattach the retina in cases with an extremely poor prognosis.

In my hands, classical types of scleral resection have been much less effective, mainly because the ridge created in the wall of the eye is neither durable enough nor located properly in relation to the retinal break.

DR. GRAHAM CLARK: This, I think, is most interesting, but I should like to point out that there is an essential point of difference, in that, as I pointed out originally, the scleral shortening procedure to which I was referring was intended to create a simple detachment from a complicated one. It was based on the assumption that the present treatment of simple detachments was adequate.

I perhaps came to surgery of the eye through improper channels; I came in as a general surgeon, and therefore I find it a bit frightening, first, to contemplate the buckling operation in its creation of what we should term a dead space, and, second, to complicate the creation of the dead space by the implantation of a foreign body.

It is interesting to note that the full-thickness scleral buckle rejects the foreign body and resumes its normal form, demonstrating the tendency of all tissues to resume their normal position, and yet the lamellar buckle is said not to. Could Dr. Schepens tell us whether there is any evidence of a return to normal form when the buckling is done in the more usual area?

The question of the postoperative period I shall have to save until later, because they have asked me a question on that and I cannot let all my powder go at once.

DR. DONALD M. SHAFER: I feel like a good Republican at Truman's birthday party! Here I am surrounded by three ardent sclerectomists. Actually, I am very much interested in Dr. Schepens' handling of the macular hole. There is a

question coming up on it later, and I should like to add something to it. More directly, I wonder what is the fate of the retina that has been abandoned. What happens to the retina that is anterior to this buckle?

DR. JAMES S. SHIPMAN, Philadelphia: I would like to ask Dr. Schepens what happens to the vortex veins when he does this procedure well back of the equator. If he does a sclerectomy, and from what I observed from his pictures he does a laminated sclerectomy, as most of us do it, and puts a polyethylene tube in the bottom of it besides, giving a nice buckle, it would be safer, in my opinion, to use some Walker pins back there, rather than a resection with these tubes. As you all know, the vortex veins come in 5 to 9 mm. back of the equator, and there are four, and sometimes five, of them; in taking out a piece of sclera 4 mm. wide and 30 mm. long, one is bound to encounter some of them. I think it is more advantageous to use the diathermy tip to obtain coagulations without cutting the sclera.

DR. CHARLES S. SCHEPENS, Boston: Scleral buckling creates an abnormal shape of the globe that is designed to reattach the retina as rapidly as possible. Methods of buckling tested so far permit one to create a ridge in the fundus that will stay from one to six months or more, depending on the technique used. It is possible that no method of scleral buckling designed so far produces a permanent ridge in the fundus. In practically all cases the tube is slowly pushed outward by the intraocular pressure. It is not extruded from the tissue but remains imprisoned under the scleral sutures, and becomes surrounded by new-formed connective tissue. Extrusion has been observed in less than 1% of the cases operated upon and is the result of faulty technique. The flattening of the ridge, after several months, is rarely followed by a relapse of the detachment. The latter occurs only in particularly unfavorable cases, and special techniques have been developed for creating a buckle lasting at least six months in such cases.

Sections made of human specimens after this procedure show that the reaction caused by the polyethylene and the suture material is very mild.

The retina which is not treated and is anterior to the buckle remains detached, and this seems to cause no trouble to the patient.

It is true that in many cases the buckle is located either over a vortex vein or over its ampula. With a proper technique this can be safe. A subchoroidal or vitreous hemorrhage is generally caused by pricking or tearing a venous channel, and this we try to avoid carefully. When necessary, one or even two vortex veins may be safely tied.

DR. JOHN M. MCLEAN: Dr. Shafer, what are the rationale and indications for the use of vitreous in retinal detachment?

DR. DONALD M. SHAFER: First, I want to explain what we are using and calling vitreous. This material, which is a sterile pooled aspirate of human vitreous, is what is extracted through an 18-gauge needle from eyes coming into the Eye Bank for corneal transplants. I have called the operation a vitreous implantation. This is not to be confused with vitreous transplantation, which is done in cases of hemorrhage. In implantation, we do not remove any vitreous; we are adding vitreous.

The factors which make up the rationale for the use of vitreous are basically three. The first I should call the anatomic factor. Anatomically, the retina is attached firmly at the disc and firmly at the ora. In between, it is attached only by the very nebulous processes of the pigment cells. So here we have a loosely attached structure suspended across this arc of the choroid and sclera, and the hypothesis was, and is, that the vitreous is the essential normal support to the retina in between its two strong attachmens. By the same reasoning, it would seem logical to implant vitreous to help put back retina that is detached, and keep in place retina that has been reattached. Clinically we know, for example, that the incidence of retinal detachment following cataract extraction parallels, more than probably any other single factor, the loss of vitreous.

I feel that this factor probably is operative in most or all retinal detachment, in that the subretinal fluid is probably degenerated vitreous. Therefore it represents a subtraction from the normal vitreous volume. Again, it would logically follow that implantation of more vitreous would represent more support to the retina.

The second factor in the rationale of vitreous implantation I call the surge factor, and this, of course, is not original with me. It is similar to the thinking of Lindner; and it is that after the movement of the eye, the vitreous continues to move and pull on any point where it is attached. I believe most people will agree that many retinal detachments are preceded by vitreous detachments, in which the vitreous is detached posteriorly and comes forward. It could be hypothesized, therefore, that this vitreous framework would be free to swing or pull on the remaining ora attachment in a low-viscosity medium, presumably intraocular fluid, posteriorly. It would follow, therefore, that the implantation of more vitreous, a high-viscosity medium, would tend to reduce the amount of surge, and thus the amount of traction on the retina.

The third factor in the rationale is purely surgical; that is, by implanting vitreous, one is hydraulically pushing out subretinal fluid from behind the retina through the drainage hole and further pushing the retina up against the choroidal diathermy area.

The rationale consists, then, of implanting vitreous to supply more support for the retina, to reduce the surge factor, and to drive out the subretinal fluid, pushing the retina against the choroid.

As to the indications for the vitreous implant in detachment of the retina, I can probably best show them by presenting some slides; but before I do, I want to say we have now done between 65 and 70 operations with vitreous, not all of them in treatment of retinal detachment. I want to emphasize that we have never lost an eye. We have never had panophthalmitis or sympathetic ophthalmia. We have not had worse vision after operation than we had before operation, even though we failed to attach the retina. The first slide will give an idea of the cases we were working with. One of the men at the Manhattan Eye, Ear and Throat Hospital said that we operated on everything but cases of phthisis bulbi, and there was some doubt about that. In no instance was a fresh case involved in this series. They are all what Dr. Clark called Class 2 or Class 3 cases. The cases are consecutive, and there was no selection. It must be stressed that every case was a previous failure, at least once, with an accepted technique.

The preoperative status of the 42 consecutive cases of vitreous implant for retinal detachment included 3 cases of Slaine implant plus diathermy failure; 12 cases of previous scleral resection, drainage, or shortening failure; 14 cases with three or more previous diathermy or scleral surgery failures; 16 cases of aphakic detachment; 8 cases of previous diathermy failure with fixed folded retina; 22 cases of total retinal detachments, and 8 cases with fixed vitreous bands. Most cases included one or more of the above characteristics.

The next slide shows the results in the main categories. Of the 42 cases which I said were all previous failures by an accepted technique, 16 had aphakic detachment; in 44% of these the retina was reattached. Of the 22 cases with total detachment, 37% had the retina reattached. Of 22 cases with myopia of less than 5 D., 55% had reattachment; of the 4 with 5 D. or more of myopia, 25% had reattachment. Of the eight cases with fixed vitreous bands, 50% had reattachment. These results show the indications for vitreous implant.

The criterion of success was that all available retina be completely reattached for six months or longer. I say "available retina" because in this series are cases of retinal fibroplasia in which the periphery is no longer true retina and we have reattached the posterior portion that remained as retina. Of these 42 consecutive cases, all failures by previous methods, the available retina was completely reattached by vitreous implant in 48%.

I think the best answer to what the indications are is on the slides. I want to thank Dr. R. T. Paton and Dr. H. M. Katzin for permitting me to work with the Eye Bank and providing me with vitreous, thus making it possible to do two years of animal surgery before I dared try our first human case, and Dr. Milo Fritz, who is now practicing in Alaska and who began to save vitreous, some of which is six and a half years old. I also want to thank Dr. David Webster for his moral support and his technical support when things got difficult.

JOHN M. MCLEAN: Dr. Shipman, what are the relative merits of a lamellar resection and a full-thickness scleral resection? What are the indications for each of these?

JAMES S. SHIPMAN, Philadelphia: I cannot refrain from congratulating Dr. Shafer on his excellent report of vitreous implants; his results are almost as good as or better than I obtain in the usual run of cases. Certainly the results are as good as or better than we get in resections.

Concerning the relative merits of the lamellar and the complete resection, I do not do a complete resection any more. I think lamellar resection does everything that the complete resection does, and without the dangers. In the complete resection you are apt to lose subretinal fluid before you want to, and also, as Dr. Schepens has pointed out, all eyes have a tendency sooner or later to return to normal; the sutures either slough out or break, and the sclera comes back to the position it was in originally. If this happens in a complete resection, and I have seen it happen, a marked herniation of the choroid may result, which is very embarrassing, to say the least. But with the lamellar resection this is much less apt to happen. When a thin layer of sclera is left and it has been coagulated to a certain extent, one is much more apt to get a thin fibrous coating and the scar is likely to be much firmer. For this reason, I think the lamellar resection has definite advantages, and one can let the fluid out by different ways. Most of it I get out by pulling the Walker pins out. The suture shown on the left of the slide was Dr. Patrick Kennedy's idea; when we were doing the complete resection, we were fearful of not having enough to hold on to. These pictures show that the tear has been localized, and I go well back of this area, and also surround the tear with Walker pins.

The tear has been localized by direct ophthalmoscopy and this point marked on the sclera. I completely surround the area of the tear and make a barrage of coagulation back of this. I mark off the area on the sclera that I am going to resect, and this is usually anterior to the tear. I make my scoring of this part with a Lundsgaard knife, tapering this off at each end. I then undermine the strip of sclera with the Lundsgaard knife and completely remove the outer four-fifths of the sclera. In the slide you can see in some places a little of the choroid, which is shining through. The piece of sclera to be resected is stripped off. I want to stress here a procedure which one of the residents at Wills Eye Hospital pointed out to me, and I hope I have not gotten too old to learn from a resident. Indeed, I think I learn from them more than they do from me at times.

Here we undermine the posterior edge of the sclera, and the next picture shows the anterior edge being undermined at the same level where we have cut down to the posterior layer, or one-fifth of the sclera. Here we are putting in our sutures. We use single-arm sutures, not a mattress suture, as I feel if the mattress suture breaks we lose two sutures. Before we tie our sutures, we spread the loops apart and coagulate this lower, or deeper, part of the resected area with light diathermy, using the tip of the Walker electrode, with 20 to 30 ma. of current, as recorded on our instrument, an ophthalmotherm.

We have pulled the Walker pins out here, as we are ready to tie our sutures. This lets the subretinal fluid gush out and helps the retina to float, or pop, up rather suddenly and come against the soft coagulum.

Here is where our tear is, and we are anterior to it, but we are definitely posterior to it here, and we must be. Dr. Schepens is right: You have to form a barrier back of the tear. Whether you create it with buckling or with coagulation of the sclera I do not think makes much difference, as long as you have shrunk the sclera enough to pull it with the choroid to the retina. You must have soft coagulum in the choroid back of the retinal tear, so that when they do come in contact an adhesion forms. You must keep these structures in contact long enough to insure a firm scar.

The next picture shows the rectus muscle reattached and the closing of the conjunctival wound with a continuous suture, which is very easy to remove one week later.

DR. JOHN M. MCLEAN: Dr. Clark, what are the most important factors in employing diathermy on the eye?

DR. GRAHAM CLARK: The most important factor is to know what you are doing.

The term "diathermy" refers to the heat produced by high-frequency electrical currents encountering the resistance of tissues. In treating detachments, we use

this heat to create the choroidal lesions whose adhesive reaction seals the retinal holes; but all diathermy is not suitable for this purpose, for the diathermy effect ranges from the pleasant warmth of the sinus-treating pad, without damage to the most delicate cell, to the instantaneous dissolution of tissue, where the point of contact of the electrode resembles a knife cut.

The factors producing these differences are important and must be borne constantly in mind. They may be stated as follows:

1. The heat produced is directly proportional to (*a*) the resistance encountered, (*b*) the time of application, and (*c*) the square of the current. Of these, only the time is at the control of the surgeon, for the resistance of the tissues varies widely at each application of the electrode, and the current varies inversely with the area of the electrode contact.

2. The nature of the heating depends on the frequency and intensity of the current and on the pattern of the wave train, whether "damped" (irregular), as from a spark gap, or "undamped" (uniform), as from a vacuum tube oscillator.

(*a*) The low-intensity, low-frequency, damped current boils the tissue fluid and explodes the cells with their own steam. This is the "desiccating" or "fulgurating" current.

(*b*) The high-intensity, low-frequency, damped current produces dense coagulation and carbonization. This is the "coagulating" current.

(*c*) The high-intensity, high-frequency, undamped current produces dissolution of cells with little coagulation or carbonization. This is the "cutting" current.

(*d*) The "blended" current is a combination of (*b*) and (*c*).

3. The heating effect does not necessarily occur at the point of electrode contact but takes place wherever a sufficient density of current meets sufficient resistance. We have specimens to show that this may be the bifurcation of a blood vessel as much as a centimeter from the point at which the electrode was applied. It follows, then, that the electrode employed should be as small as possible and applied as precisely as possible. The pointed electrode may be likened to the [illegible] whereas the surface-ball electrode resembles the shotgun.

4. Low-frequency currents are fickle and will always follow the path of least resistance. This may be the layer of moisture on the surface of the sclera, a blood vessel, the zonule of the lens, or the vitreous.

Today's market of electrosurgical instruments is flooded with a myriad of sleek, shiny instruments, all labeled "diathermy" and all declared to be suitable for retinal detachment work. These instruments cover a frequency range from 50,000 to 27,000,000 cycles and wave train patterns of infinite variety; added to this, tests show that the output of any two instruments of the same make differs widely in the spark-gap generator group. It is therefore important to know your own instrument.

JOHN M. MCLEAN: Dr. Schepens, what is your method of scleral depression? Are any dangers involved in its use?

DR. CHARLES L. SCHEPENS, Philadelphia: Scleral depression is more easily performed with an indirect stereoscopic ophthalmoscope, for the following reasons: The instrument has a better working distance and a stronger light than any direct ophthalmoscope; the field of view and depth of focus are considerably larger, and this helps with orientation; stereopsis is also a considerable advantage.

For pressure on the sclera I use a small instrument mounted on a thimble, and called a scleral depressor. It is very useful, although not indispensable, in dilating the pupil fully. When the upper or lower region of the fundus is to be examined, pressure may be exerted over the eyelid; when the nasal or temporal portion of the fundus is to be examined, pressure is exerted over the bulbar conjunctiva, after instillation of 0.5% tetracaine drops. Only very light pressure is required to see the ora serrata; the beginner generally presses too hard on the globe. If the intraocular pressure is markedly elevated, pressure is ineffective and should not be used; after intraocular surgery, scleral pressure should be deferred for six weeks.

No complications have been observed as a result of the correct use of scleral depression. This method appears as safe as tonometry or pupillary dilatation: Such procedures may cause complications if the proper precautions are not taken, but the information gathered by their use is of enough use that it is reasonable to take this calculated risk.

DR. JOHN M. MCLEAN: Dr. Shafer, how do you do a vitreous implant for detachment of the retina? Where do you get the vitreous?

DR. DONALD M. SHAFER: Before we get into the diathermy part of the surgery, we prepare a sclerectomy incision at right angles to the limbus, about 8 to 12 mm. behind the limbus, cut down through the sclera to the choroid, and put a mattress suture edge to edge through this incision and a second mattress suture in the opposite direction, so that there are opposing mattress sutures. We experimented with many different types of sutures and found this was the only one that would hold well at normal tension or at higher tensions. Then the operator goes ahead with the diathermy technique, whatever that may be. At the conclusion of that, and the provision for the subretinal drainage, a syringe filled with vitreous on an 18-gauge needle is put through the previously prepared sclerectomy opening, 8 to 9 mm. behind the limbus, after the choroid has been perforated by diathermy. The assistant holds the pair of mattress sutures; the eye is brought up to whatever tension is wished (I have measured it at 70 mm. on a tonometer, but normally I bring it to approximately 50 mm. and hold it for two minutes); then the pressure of the eye is permittted to drop, as it does almost automatically from the subretinal fluid loss. The assistant pulls the sutures tight; the needle is withdrawn (there is usually one drop of vitreous lost from the needle), and the sutures are tied.

(Slide) A point that we have to be particularly careful about is striking the needle against the lens. In an aphakic there is, of course, no problem. We have tried the anterior route many times and found it much less successful.

Where do we get the vitreous? The vitreous comes from Eye Bank eyes. This is sterile pooled aspirated human vitreous. The average eye will produce approximately 2 cc. of vitreous. It is secured by piercing the seared sclera of the donor eye at about the equator and aspirating the vitreous with a sterile 18-gauge needle on a syringe. The vitreous is kept in a household refrigerator at 40 F in rubber-capped glass vials.

DR. JOHN M. MCLEAN: Dr. Shipman, under what circumstances do you advise against operation for retinal detachment?

DR. JAMES S. SHIPMAN, Philadelphia: When the patient does not want it, primarily. These are not many, but if the patient does not want surgery, I will not operate. That the patient wants it and has only one eye would make a difference. The presence of diabetes with diabetic retinitis proliferans would influence me not to operate. I have always made it a practice to tell the patient the truth, as far as I know it, in regard to what is going to happen if he does not have the operation, how long he will be in the hospital, and how long he will be away from work, with a discussion of the expense of the entire procedure; if after he knows all the facts, he still wants the operation, I will do it.

JOHN M. MCLEAN: Dr. Clark, in reoperating on a patient, how would you handle a sclera which has become partially necrotic from previous surface coagulation?

DR. GRAHAM CLARK: After either surface or penetrating diathermy, the sclera in the area of the treatment will not take sutures for three months. If a scleral shortening operation is deemed necessary, it must be done in another area. If vitreous traction is the causative force, the pole opposite the hole is usually the area of choice. If shrinkage or a constricting membrane is the main cause of failure, the area adjacent is usually best.

DR. JOHN M. MCLEAN: Dr. Schepens, what is your technique for localizing tears during the operation?

DR. CHARLES L. SCHEPENS, Boston: For the purpose I use exclusively indirect stereoscopic ophthalmoscopy; some of the advantages of the method were described in my answer to Question 6 (What is your method of scleral depression?). The

low magnification obtained in indirect ophthalmoscopy helps one to see through hazy media and to the extreme fundus periphery; the disadvantages of low magnification are offset by strong illumination.

For localization of a retinal break, the surgeon retracts the soft tissues from the globe in the quadrant under examination; the observer places the break in the middle of his ophthalmoscopic field; this creates a red glow in the corresponding area of the sclera; the surgeon applies a flat diathermy electrode (which has an area of 1 sq. mm.) over the sclera, in the middle of the red glow; as the electrode is pressed firmly against the sclera, the observer sees a choroidal indentation and directs the surgeon as to exactly where the electrode should be applied; when the electrode is in the correct location, the diathermic current is released. A white mark will form in the retina only when the detachment is flat.

DR. JOHN M. MCLEAN: Dr. Shafer, what is the importance of letting out fluid in retinal detachment surgery? What is your technique for this procedure?

DR. DONALD M. SHAFER: I can see only one reason for letting the fluid out, and that is to permit the retina to go back against the choroid, or, at least, against the area of choroidal reaction.

As to the technique, I use one of two methods, depending on how far the retina is from the choroid. As Dr. Schepens and Dr. Clark mentioned, the great danger is in creating a new hole in the retina by the method you use to get out the fluid. Therefore if the retina is flat, I usually make a tiny sclerectomy incision down to the choroid, until the choroid begins to herniate, and then open it with diathermy. One should try to have the choroid bulge externally rather than allow the tip of the electrode go in. If, however, the detachment is a large bullous one, I use pins, taking care that the tip of my pin does not touch the retina. The one advantage of pins is that they save time.

DR. CHARLES L. SCHEPENS, Boston: How do you know, when you use pins, that you are releasing the subretinal fluid, and not fluid vitreous escaping through new holes made in the retina by the pins?

DR. JAMES S. SHIPMAN, Philadelphia: You can cause a tear, and you can certainly close it, when you see you have made one with a pin.

DR. CHARLES L. SCHEPENS, Boston: How do you know, when you operate in a case, that you are going to be successful? I have seen cases in which 10 or 15 additional retinal holes were caused by pins. If such a case is going to be successful, additional holes are unimportant; if it is not, they greatly complicate reoperation because such holes are always very small and difficult to detect.

DR. JAMES S. SHIPMAN, Philadelphia: If the retina has flattened out, we do not put in pins; we use surface coagulation. I am talking about the big bullous detachments. I still feel that the Walker pin gives the best result with a resection in that type of case.

DR. GRAHAM CLARK: There is one possibility by which both can be right, and that is by the injection of air, helium, argon, or vitreous. This reduces the possibility of having a hole in the retina which is not in contact with the exudative area. We have found that closure by this exudate is complete within 36 hours, and any of these injected substances will hold the retina against the exudative area for that time.

DR. JOHN M. MCLEAN: Dr. Shipman, how would you handle a detachment with a macular hole?

DR. JAMES S. SHIPMAN, Philadelphia: Personally, I have not had many to handle, and the detachment does not occur very often when it is a macular hole alone; usually we have other holes. All of us have seen so-called macular holes dozens of times which never lead to detachment, and it is very fortunate that they do not. I have always felt that most of these so-called holes are cysts. When a detachment does develop, I would treat it like any other type, except I do not use quite as much coagulation or current. You cannot expect to get good results in these cases, and that is true in many detachments in which the macular area is involved. I always tell a patient over 50 years of age that he will never have

reading vision when there has been a detachment of the retina in the macular area for over three days. In the three macular holes I have had the opportunity to treat I have used coagulation. Some men have advocated treatment with electrolysis or the galvanic current, or use of the undermining method with potassium hydroxide, as advocated by Lindner; but this is difficult to control. I have used the last method, but not in macular holes. I would like to know what Dr. Schepens thinks.

DR. CHARLES L. SCHEPENS, Boston: I completely agree with Dr. Shipman's statement that macular breaks are extremely rare; I found them present in about 1% of all cases of retinal detachment. In many instances a macular break is mistakenly diagnosed and operated upon when only a macular cyst is present. In a case of retinal detachment with peripheral breaks in which a macular break is suspected, I treat the peripheral breaks only. The suspected macular break is treated in a second operation if this proves necessary.

DR. DONALD M. SHAFER: It is difficult to determine the macular situation. With a borderline macular picture the peripheral tears are taken care of by diathermy, and if a detachment is not reattached, we feel a macular tear should be assumed. We used a thermophore at 160 F for one minute, as described in the thermophore manual, and we had two excellent results, not quite 20/40; but had 20/70 in one eye, which has slipped now to 20/200, and 20/200 in the other. It is an interesting use of the thermophore to produce a very soft choroidal reaction; it is not as intense as that we usually get from diathermy.

DR. JAMES S. SHIPMAN, Philadelphia: This method was described by Langdon, of Philadelphia, about 15 years ago. I think he reported 9 successful results out of 11 cases treated by this method.

DR. JOHN M. MCLEAN: Dr. Clark, what are the relative merits of the use of multiple pins versus that of a single electrode?

DR. GRAHAM CLARK: The older, low-frequency, fickle currents require the use of micropins for penetrating application, for they allow multiple applications to be made without the escape of subretinal fluid, which would dissipate the current. They also keep the globe firm, allowing use of the force necessary to penetrate the sclera with these currents. Another point is the ease with which they can be visualized within the globe, insuring the proper location of the treatment ring. They also leave a patent drainage hole for the subretinal fluid, which the dense coagulum would otherwise plug.

Their disadvantages lie in their usual length, so that the tip, where the current density is greatest, passes the choroid, where the reaction is desired, and may damage the retina. Further, the sudden removal of multiple pins is accompanied by a rush of fluid, which sucks the retina into folds and does not allow it to settle smoothly.

With the more modern current the coagulum formed is not sufficient to hold the pins in place, and they will drop out when released.

These higher-frequency currents are not disturbed by surface fluid; so the single 0.5 mm. electrode is best, allowing adequate and widespread, slow, prolonged drainage and not endangering the retina.

DR. JAMES S. SHIPMAN, Philadelphia: Don't you feel that when you get 10 to 15 points squirting at once, you are going to get a vacuum, which is likely to pull the retina to the coagulated areas in the choroid?

DR. GRAHAM CLARK: I think this will pull the retina in place, but I believe you might better say "jerk" it in place.

DR. DONALD M. SHAFER: With the technique we use we do not have to worry about "jerking" or "squirting," because the subretinal fluid can be driven out according to the amount of vitreous we put in.

DR. JAMES S. SHIPMAN, Philadelphia: You push it out with pressure from within.

DR. CHARLES L. SCHEPENS, Boston: There is one disadvantage common to all methods of injection into the vitreous cavity. The size of the needle used for the

injection is necessarily such that episcleral tissue will later grow into the vitreous through the opening made in the sclera by the needle. Months or years later this connective tissue ingrowth may cause an incurable type of retinal detachment.

DR. JOHN M. MCLEAN: How does your biomicroscopic examination of the vitreous influence your treatment of a case, Dr. Schepens?

DR. CHARLES L. SCHEPENS, Boston: In the majority of cases, biomicroscopic examination of the vitreous does not influence my surgical procedure. This examination only helps to explain certain features of a retinal detachment. For instance, fixed retinal folds are sometimes caused by traction bands or membranes in the vitreous, which cannot be seen by any other means of investigation. It is true that there are cases with fixed retinal folds which show no visible band or membrane in the vitreous cavity.

DR. JAMES S. SHIPMAN, Philadelphia: Then you give a patient a poor prognosis when you find such folds?

DR. CHARLES S. SCHEPENS, Boston: Yes.

DR. JOHN M. MCLEAN: Dr. Shafer, what criteria do you use to determine that reoperation is necessary, and how soon after an unsuccessful operation do you reoperate?

DR. DONALD M. SHAFER: If after surgery I find, first, a new tear that was not incorporated in my diathermy and a detachment of the retina, it is obvious I have to reoperate. Second, if the retina is detached and the area of choroidal reaction is very thin, the operation is a failure and reoperation is necessary. In borderline cases, where there is a small detachment in the periphery which has been surrounded by diathermy, with the tear included within it, but the field starts to change, there is obviously a leak and that means reoperation. To me an important factor is the status of the other eye. If the other eye is blind, there is no choice about what you must do in a case of detachment. If, however, the patient has had four diathermies and two or three other procedures and has spent most of the last year in the hospital, and so on, then sometimes I do not reoperate if the other eye is serviceable. I think, as Dr. Shipman said, that a tremendous amount depends on the patient's decision as to whether he wants another operation.

When to operate I believe depends on an evaluation of the patient's general condition and the status of the macula in the eye. If the patient is debilitated from continued surgery and the macula is not threatened, I may be able to postpone operation until the patient has been built up generally. If, however, the patient is in good condition and the macula is threatened, then I operate as soon as I can.

DR. JOHN M. MCLEAN: What is your preoperative management of cases of retinal detachment, Dr. Shipman?

DR. JAMES S. SHIPMAN, Philadelphia: First, we have to make the diagnosis of detachment—find the tear if we can; we cannot always find it. Certainly, we have found it lately more frequently than we used to. After we have found the tear, we tell the patient the whole story—what he is up against; what we think should be done—and we tell him that surgery is the only thing for his particular condition. If the patient agrees, we send him to the hospital and put him in bed. I think I keep my patients longer in the hospital than some of the others here. I order pinhole goggles with 3 mm. openings to be worn during the day. We put the patient in a position that encourages gravity to help the detachment to settle back in place. We keep the pupil dilated with atropine or, if necessary, use other mydriatics, and we keep studying the eye ophthalmoscopically to see what is taking place, and look for the folds Dr. Schepens is talking about, not being satisfied with finding one tear, but looking for others. We have the patient prepared for general anesthesia. We formerly used 2% procaine retrobulbarly, but I have been criticized for it. Now we practically always do our detachment operations with intravenous thiopental (Pentothal) sodium anesthesia and keep the patient in bed, in a favorable position, for five days. If the patient does not show any signs of the detachment going back, we do the operation, usually a resection. If the

retina goes back, we are happy and feel there is a better chance for a successful result. These cases require a less formidable procedure.

Dr. Graham Clark: Our preoperative regime is based on three concepts:

1. A patient cannot be physically quiet unless he is mentally quiet.

2. A patient cannot be physically quiet unless he is comfortable.

3. The high-speed movements of the eye are the cause of the elevation of the retina and can best be stopped by firm, opaque bandages.

We therefore first give the patient a 12-hour period of bed rest without any eye cover (usually over the first night), so that he may get used to his surroundings and become familiar with the people who will care for him and see the manner in which things are done. During this time every step of the procedure through his convalescence is explained in detail.

After this the eyes are bandaged and the patient is asked to lie quietly, with the Gatch bed elevated to 30 degrees and slow turning of the head allowed. After 24 hours the eyes are again examined and the bandages reapplied. Prior to the operation a final examination is done.

We have not found that the patient's position in relation to the position of the retinal elevation has any bearing on the speed of settling.

Dr. Charles S. Schepens, Boston: After admission to the hospital a complete examination of both eyes is performed; this includes visual fields, tonometry, slit-lamp examination, and thorough fundus examination with the indirect stereoscopic ophthalmoscope. A detailed fundus sketch is made and checked by at least one other observer. A medical check-up is also performed, and it always includes a chest x-ray. The patient is neither given pinhole glasses nor put to bed, except when there is immediate danger of macular detachment. No preoperative flattening of the detachment is desirable before a buckling procedure. On the morning of the operation the patient is made to walk until he receives his preoperative sedation.

Dr. Donald M. Shafer: I put my patients to bed with pinhole glasses until I am convinced that we have the whole story from studies of the fundus. I think an important thing in the preoperative period is the other eye. We look at it, too. For dilatation I prefer 5% to 10% homatropine and 10% phenylephrine (Neo-Synephrine) hydrochloride three times a day, and the rest is essentially what has been said.

Dr. John M. McLean: Dr. Shipman, what is your schedule for postoperative management in the hospital and after the patient is discharged from the hospital?

Dr. James S. Shipman, Philadelphia: This is where I am going to get stepped on! I do not dress the patient's eye for 48 hours after surgery, and I dress it and instill atropine daily after that and keep the patient in the same position he was in before surgery. At the end of a week I take the conjunctival sutures out. At the end of two weeks I give the patient the same pinhole goggles he had before operation, but still keep him in the same position, and at the end of three weeks I start to get him up. I do this very gradually. I have had the experience of seeing more hemorrhages from getting the patient up too fast than from giving too much diathermy current. The day before the patient leaves the hospital I give him a 6 mm. opening in the goggles, and within two weeks he gets a 12 mm., and in a month a 24 mm., opening; and after another two to three weeks he gets a modified type of goggles. These are shown in slides and have been described by us elsewhere. With these the patient can look at television and do a few things around the house. We gradually remove the black enamel from the back of these goggles but leave the temple guards on for another month. The patients usually do not want to take them off; they really get to like them. We feel that after six months these patients are safe, and we then allow them to follow their usual routine and line of work; however, we have them report to the office every three months for a year and then check them every six months for a year or so, and then every year as long as they will come. How one can take a chance in these cases I cannot understand. I have never had a patient object to waiting another month or two to get back to work if he is getting a good result. I am happy, and so is the patient; and I think it is worth the effort.

DR. CHARLES L. SCHEPENS, Boston: The postoperative management after scleral buckling differs from that following retinopexy. At the end of a scleral buckling operation, 200,000 units of penicillin, dissolved in 1 cc. of water, is injected into the orbit. Mydriatic drops are instilled and a pressure dressing is applied for 24 hours, in order to avoid excessive swelling of the soft tissues. The dressing is renewed daily, and a fundus examination is performed every day. In favorable cases the patient is given dark glasses and gets out of bed in 4 to 6 days; he is discharged from the hospital 8 to 12 days after operation and is kept on mydriatics for 2 more weeks; he is then allowed to return to work gradually.

More caution should be exercised in mobilizing a patient after retinopexy; it is generally not safe to let him out of bed for 12 to 14 days.

DR. DONALD M. SHAFER: We do not do the first dressing for 4 days after operation; then the eyes are dressed at 4- to 5-day intervals until the 14th to 18th day, or whenever the choroidal reaction has ceased, at which time, if the operation is successful, the patient is given pinhole glasses and gotten up by the 21st day. He is kept in pinhole glasses for approximately another month, the pinholes being enlarged as Dr. Shipman spoke of. This is a tremendous psychological advantage to the patient; in walking on the street, he is not pushed around as they are in New York; the black glasses are good as a warning to other people, and I think they keep some retinas on.

DR. GRAHAM CLARK: Our present schedule of postoperative care differs rather from the more generally held concept. It arose from the careful analysis of many hundreds of cases as to what were the essentials. It has been gratifying to find that in the three years and the over 300 cases in which this schedule has been tried our percentage of successes has risen and our percentage of recurrences has decreased.

In essence, we believe (1) that the success or failure of the treatment has been determined when the patient leaves the operating table; (2) that the chief force effecting the separation of the retina is the inertia of the vitreous in the movements of the eye, and (3) that the patient who rests in a comfortable position and moves slowly will be quieter than one who is tortured by forced immobility in an uncomfortable position.

The resulting schedule is as follows:

1. At the end of surgery both eyes are bandaged and a protective mask is applied.

2. Until he has reacted from his anesthetic, the patient is placed on the side which will allow the air bubble (injected into the vitreous space) to ride against the area of diathermy treatments.

3. After recovery from anesthesia the patient is allowed to lie quietly as he chooses and have a Gatch bed elevated to 40 degrees. He is instructed again in slow movements, but encouraged to exercise in this type of movement.

4. On the second postoperative day the dressings are changed and the patient is allowed to be up in a chair as desired and is allowed lavatory privileges. He may be shaved and may brush his teeth and comb his hair.

5. On the sixth day the dressings are changed for 5 mm. pinhole goggles and the patient is allowed to walk about.

6. He is allowed home on the 10th to the 14th day. He is instructed simply to lead a quiet, convalescent life, with the avoidance of anything causing him to strain. He wears his pinhole glasses all the time his eyes are open but sleeps without a mask. Atropine 1% is instilled daily in the eye operated on.

7. He is examined every two weeks, and the pinhole glasses are removed at the end of a month.

8. During the second month he gradually increases his activity to reach his normal routine by the end of the month.

9. The final examination is done at the end of three months, and check-up examinations are done thereafter at six-month intervals. Both eyes are dilated ad maximum at all examinations.

Not only has this regime improved our surgical results, but the physical and mental status of our postoperative patients has improved so as to make the whole change seem worth while from that aspect alone.

DR. JAMES S. SHIPMAN, Philadelphia: You are not allowing the patient to help cure himself. I feel also that there is a certain amount of subretinal fluid left in the eye. I do not think we have to get it all out at the time of surgery; some of it will gradually seep out, and with the patient lying in bed in a position that will help it to settle, in two weeks the retina usually will be flat, even though it was elevated for a week or more after operation. I would not get the patient out of bed under three weeks if the retina is still elevated. Perhaps all yours are flat, Dr. Clark.

DR. GRAHAM CLARK: If you inject air, you will have a flattened retina at the end of the operation. My only defense to the charge that the patients will not do as well is that they seem to.

DR. CHARLES L. SCHEPENS, Boston: There is one postoperative complication which was not mentioned, namely, death following a circulatory accident caused by bed rest. Therefore, anything that can be done to shorten the period of bed rest is of extreme importance.

DR. JAMES S. SHIPMAN, Philadelphia: We have had well over 600 patients who have lain in bed for three weeks or longer, and we have yet to have a death. I have had but one case of pneumonia, and I think that is a good record. More patients have died from getting up too soon. I have had them die after cataract operation on getting out of bed, but I have not had them die in bed. I do not think the bad thing is the physical effect; it is the financial loss to the patient. Beds today are very scarce, but I do not feel this should influence the handling of these cases.

DR. CHARLES L. SCHEPENS, Boston: Out of nearly 3,000 operations for retinal detachment performed since 1947, we have recorded 5 deaths resulting from pulmonary infarction; in each case it was felt that prolonged bed rest was a very important etiologic factor. Residents in other eye departments have reported to me 12 additional instances of death from this cause.

DR. JAMES S. SHIPMAN, Philadelphia: We must have better internists in Philadelphia than you have in Boston, and we have them observe and follow our cases quite carefully.

Obituaries

JOSEPH I. PASCAL, M.D.
1890-1955

Joseph Irving Pascal was born in Kovno, Lithuania, on March 28, 1890. He died in New York City on April 22, 1955, at the age of 65. He was brought to this city by his parents as a child, was educated in its public schools, and received a B.S. degree from Columbia University in 1912. The next two years, he studied optometry at Columbia, soon turning toward the study of medicine at the College of Physicians and Surgeons, in New York. In 1913 he founded the American Institute of Optometry and, serving as its director from 1913 to 1927, launched himself on a career of teaching physiologic optics, the branch of medical science which brought him distinction and remained his main interest throughout a long professional life.

In 1927, Dr. Pascal received an M.A. degree from Columbia University and left for Vienna to resume his studies of medicine. He received his degree of Doctor of Medicine in Vienna in 1930 and spent another year as intern at the First and Second University Eye Clinics in that city.

Returning to the United States, he moved to Boston and served as chief of the refraction department of Beth Israel Hospital from 1931 to 1933. He then moved back to New York and started out in the private practice of ophthalmology, from which he retired in 1953. He served as Adjunct Ophthalmic Surgeon at the Hospital for Joint Diseases from 1934 to 1938 and at Beth David Hospital from 1938 to 1940; he was also Associate Ophthalmologist at the Harlem Eye and Ear Hospital from 1935 to 1938, and from 1938 until his death he was Director of the Eye Department at Stuyvesant Polyclinic.

In later years, Dr. Pascal turned more and more away from practicing and toward writing and teaching. He became Associate Ophthalmologist and Lecturer in Ophthalmology at the New York Polyclinic Medical School and Hospital in 1948, at the Brooklyn Eye and Ear Hospital in 1952, and at Montefiore Hospital in 1953; he also was connected with the U. S. Office of Education during World War II as lecturer in physiologic optics.

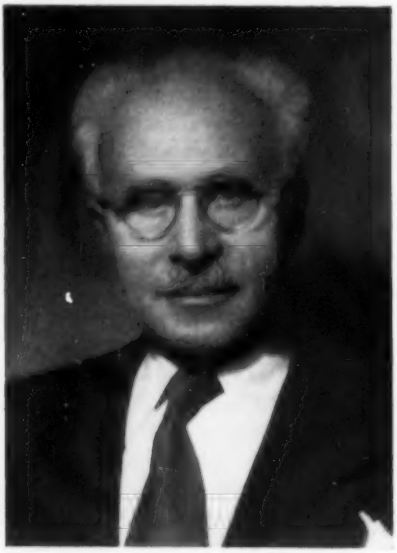
Dr. Pascal was an enthusiastic lecturer and a prolific writer. He presented papers before many of the leading ophthalmological societies—in this country; in England, France, Switzerland, and Austria, and in a number of South and Central American countries. He was especially active in the Pan-American Ophthalmologic Association, attended most of its conferences, and was appointed chairman of its Committee on Physiologic Optics. He was to have made a report at the next Congress, in Santiago, Chile, in 1956.

Of his many exhibits, the best remembered are the one on the Pascal Muscle Schema, or Benzene Ring Muscle Schema, in London, 1950, and his last exhibit on the Improved Raubitschek Arrow Test, in New York, in 1954. His exhibit on

Ocular Torsion, which he was to prepare for the 1955 meeting of the American Medical Association, in Atlantic City, he did not live to finish. The ideas he wanted to present occupied his mind even in the last days of his short, but fatal, illness. The material for this exhibit was assembled by his most faithful collaborator, his wife, Charlotte Pascal, and by their friends and served as the most fitting tribute to the memory of Dr. Pascal.

He was quite a linguist. He wrote and spoke Spanish, Italian, French, and German; helped his many friends from abroad to prepare their papers for publication in English; abstracted the Spanish ophthalmological literature for the *American Journal of Ophthalmology* and for *Ophthalmologic Literature*, and translated the proceedings of the Madrid Ophthalmological Society.

He invented, developed, or improved several instruments useful for examination of the eyes, such as the Pascal Statoscope, the self-setting cross cylinder, the



JOSEPH I. PASCAL, M.D.
1890-1955

Turville-Pascal Dynascope, and the Raubitschek-Pascal Arrow Test, and wrote several books, of which his monumental "Studies in Visual Optics," published in 1952, will probably remain a classic. Dr. Pascal's last published book (with Dr. H. G. Noyes) was the "Optometrist's Handbook of Eye Diseases," published in 1954.

Dr. Pascal's unique place in American ophthalmology was determined by the fact that he remained an optometrist as well as an ophthalmologist all his life, proving by his own example that loyalty to one group does not mean antagonism to the other. For many ophthalmologists he will remain the symbol of the perfect teacher. With kindness, humor, and perseverance, he put life into a subject which a great many of them disliked, or at least found lacking in interest.

He had students from every corner of the world. To the very end, he gave courses in refraction and physiologic optics, and many would have never passed the

examination in physiologic optics of the American Board of Ophthalmology had it not been for Dr. Pascal, a teacher with the ability to make the most complicated subject understandable.

With all his excellence, his humility remained great. There is no better summing up his attitude toward his work and students than a quotation from his Preface to "Studies in Visual Optics":

> A sage of old has said, "And lo, I have gathered wisdom from all my disciples." Every teacher will subscribe to the truth of this. And so I dedicate this work to the men and women, undergraduate and postgraduate, optometric and medical, who have come to me for counsel and instruction, and who have inspired me to search for more efficient methods, for new ways and new devices to make their studies easier and more meaningful and, in a word, to justify their faith in me.
>
> ARTHUR LINKSZ, M.D.

News and Comment

PERSONAL NEWS

Dr. William L. Benedict Honored.—Dr. William L. Benedict, Executive Secretary-Treasurer of the American Academy of Ophthalmology and Otolaryngology, was honored by his Alma Mater, the University of Michigan, when the degree of Doctor of Laws was conferred upon him at the University commencement, Ann Arbor, Mich., June 11, 1955.

Dr. Frederick H. Verhoeff Honored.—The University of Louisville conferred an honorary degree of Doctor of Science upon Dr. Frederick H. Verhoeff, Consulting Chief of Ophthalmology at the Massachusetts Eye and Ear Infirmary and Professor Emeritus of Ophthalmic Research at the Harvard Medical School, on the occasion of its commencement exercises, June 5, 1955.

Appointments in Ophthalmology, University of Pennsylvania.—The Dean of the Graduate School of Medicine of the University of Pennsylvania announces the appointment of Dr. Edmund B. Spaeth, Professor of Ophthalmology and Chairman of the Department of Ophthalmology, as Professor Emeritus of Ophthalmology. The Dean also announces the appointment of Dr. Irving H. Leopold as Professor of Ophthalmology and Chairman of the Department of Ophthalmology of the Graduate School, succeeding Dr. Spaeth.

Books

The Biologic Effects of Tobacco. Edited by Ernest L. Wynder, M.D. Price, $4.50. Pp. 215, including index, with 1 illustration. Little, Brown & Company, 34 Beacon St., Boston 6, 1955.

To those who wish a comprehensive review of the evidence for and against smoking as a calculated risk to health, this book can be heartily recommended. There are many who still believe that tobacco is not an etiologic factor in any disease save possibly thromboangiitis obliterans. Statistical evidence from highly reliable sources, however, points toward the causal relationship between tobacco and cancer and some forms of cardiovascular disease. In spite of this evidence, the manufacturers of tobacco insist that smoking is harmless, although some rather naively, as Dr. Garland points out in his foreword, "advertise the use of filters to remove substances the toxicity of which they are unwilling to admit."

The chemistry and pharmacology of tobacco are thoroughly covered, and the evidence for the production of disease states in the cardiovascular system and the gastrointestinal tract is well documented, along with the influence of tobacco in the causation of cancer. A separate chapter deals with allergy, and the final chapter covers cause and effect. There are no direct references to any ocular disabilities, for example, tobacco-alcohol amblyopia.

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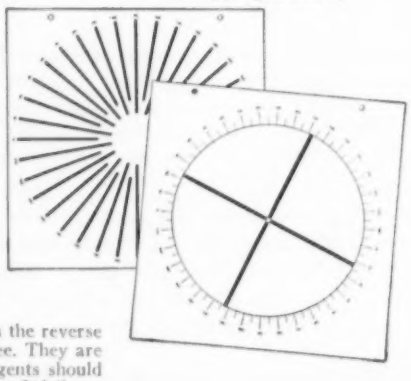

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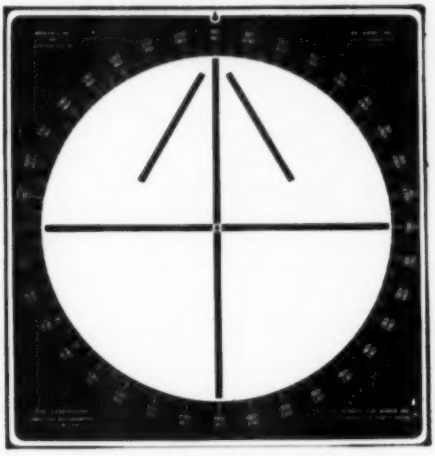

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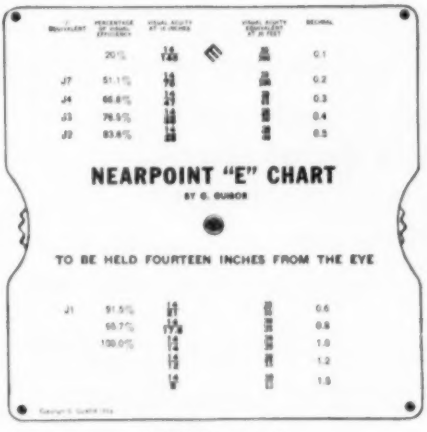

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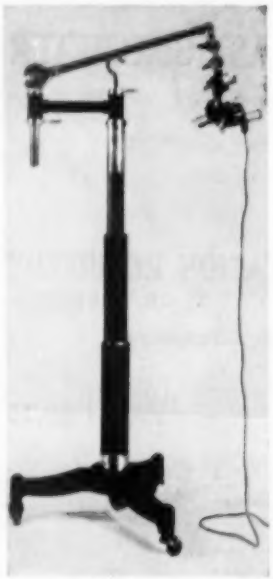

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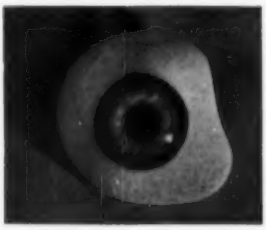

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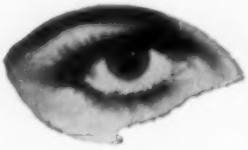

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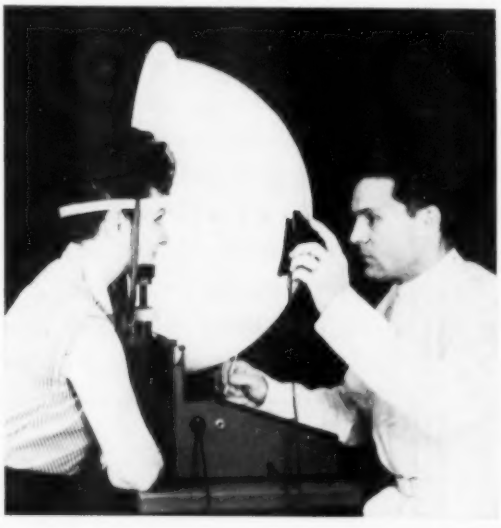

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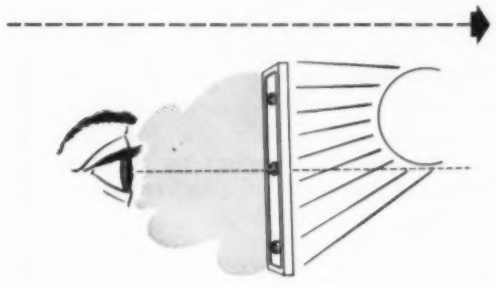

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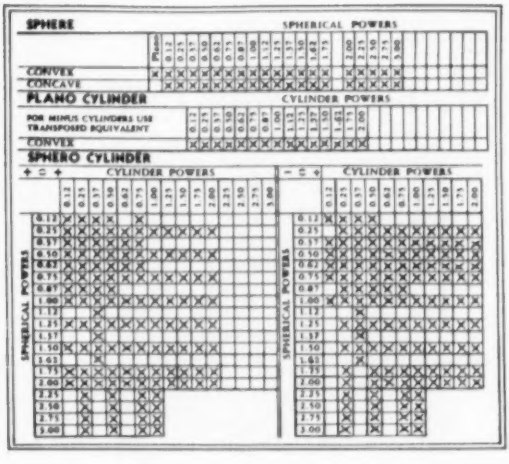

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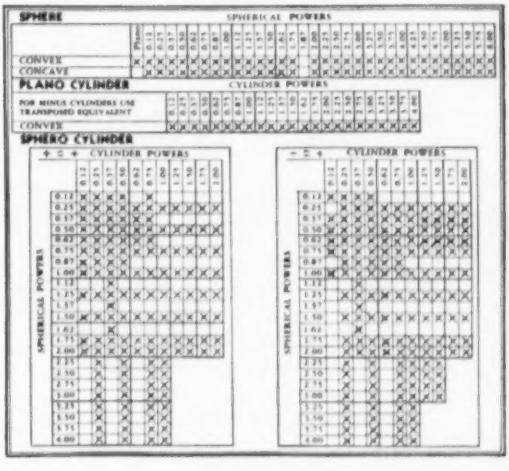

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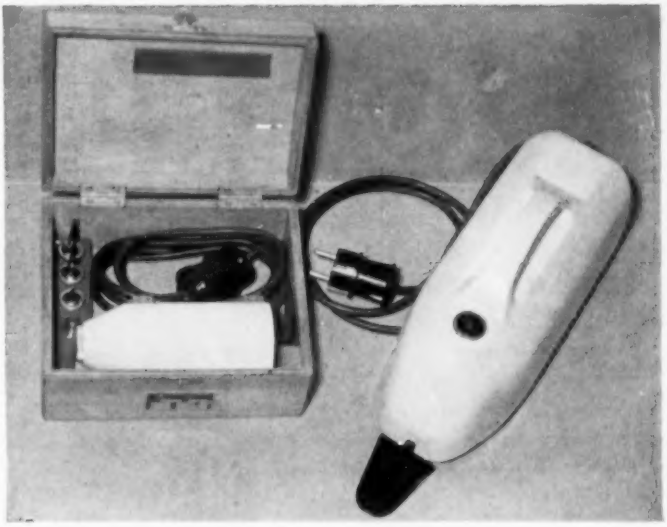

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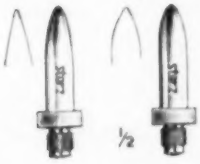

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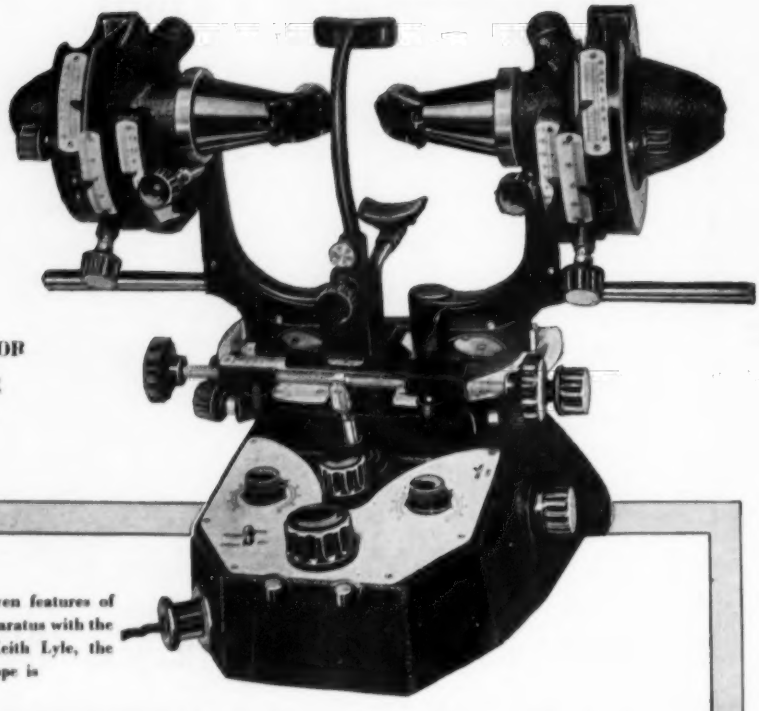

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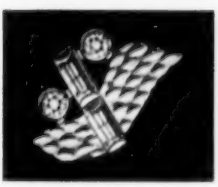

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